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PARENCHYMATOUS SYPHILITIC KERATITIS AND SYPHILITIC ATROPHY OF THE OPTIC NERVE TREATED WITH SULFOSIN*

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IT is pernaps strange to see two such widely different diseases as syphilitic parenchymatous keratitis and tabetic atrophy of the optic nerve put together, but however different are the symptoms and appearances of these diseases, they have one feature in common: though they are so closely related to syphilis, they are but little influenced by ordinary anti-syphilitic treatment with bismuth, mercury and neoarsphenamin. With the appearance of v. Jauregg's malaria treatment of neuro-syphilis, it was, however, thought that a good remedy for tabetic atrophy of the optic nerve had been found, and it was also attempted to treat syphilitic parenchymatous keratitis with malaria (Ambler, van Cleve, Schreiber, Meller, Michelsen), but the results were not very encouraging, and the statements of different authors disagreed. There is, however, a great difference between atrophy of the optic nerve and parenchymatous keratitis, the prognosis of the former being bad, whilst—as Duke-Elder says—parenchymatous keratitis is " of a chronic course and benign tendency," and exactly this difference in prognosis has determined my view on the malaria treatment of these

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two diseases. In atrophy of the optic nerve any treatment, though in itself not quite without danger, is justified, if only it is a good treatment or even only the best treatment so far; but in the fairly benign parenchymatous keratitis (Dalsgaard-Nielsen has found that this eye disease only invalids 20 per cent. totally or partly) one should take care that not " le remède est pis que le mal," and this is according to my opinion the case if parenchymatous keratitis is treated with malaria. To me it seems very questionable to inoculate a new disease—which can only partly be controlled—in order to influence a disease, the tendency of which is benign, especially when it is kept in mind that parenchymatous keratitis is a disease of children or young individuals (Dalsgaard-Nielsen: 53 per cent. between 6 and 15 years), it thus being children who for the rest of their lives perhaps must fight both congenital syphilis and malaria. To this may be added that treatment with malaria has caused necrosis of the cornea, followed by phthisis bulbi, and herpes corneae, leading to enucleation (Meller, Schreiber). Lastly, treatment with malaria should be carried out in a closed department, whilst parenchymatous keratitis decidedly belongs to an eye department.

At the time—Spring, 1933—when I started treating parenchymatous keratitis with sulfosin, I did not know of any other results of this treatment, but I have since learnt that in 1932 Schroeder had published 4 cases of parenchymatous keratitis which improved and cleared during treatment with sulfosin, according to the attending eye-specialist (O. H. Saunte) even to an unusual degree, and in the same paper Schroeder referred to a letter from L. W. Harrison, who had treated 2 cases of interstitial keratitis, one of which in particular cleared more quickly than he had seen before. This is all the existing literature on sulfosin treatment of parenchymatous keratitis.

From days of old sulphur has been regarded as a good antisyphilitic remedy and v. Jauregg places it on a level with mercury and bismuth. Sulfosin is sulphur sublimatum subtilissime grated into olive oil according to the method of Schroeder. The preparation contains 1 per cent. partly dissolved sulphur, and the non-dissolved part is present in a dispersed condition, partly microscopic. By heating, all the sulphur may be dissolved, and the preparation then has a stronger effect—but on cooling the sulphur again crystallizes out, in which case it should always be reheated before use. Treatment should be commenced with small doses (1-1½ c.c.) in order to test the sulphur sensitiveness of the individual, and the preparation is injected into the gluteal region;

* In order to decrease the inconvenience of the preparation, a new preparation, neosulfosin, has been made, containing anaesthesin, and with all the sulphur dissolved (½ per cent.); another preparation is sulfosin fortior (2 per cent.)

the largest doses given are 10 c.c., the aim of the treatment being a rise in temperature to 39-40°C.; this is, however, generally obtained with 5-6 c.c.; on the other hand, it is necessary to give such doses that the temperature really rises, otherwise there is no effect. In one of my cases—one of the earliest—it seemed that small doses, and consequently only slight rise in temperature, caused a sudden spreading of the keratitis to the parts of the cornea that had so far been free; and in two other cases a single injection of sulfosin, in spite of previous good rises in temperature, only caused a very moderate rise (38·3°C.) and simultaneously lacrimation and photophobia increased; the next injection caused a good rise in temperature, and irritation phenomena did not appear. It seems to me that this sudden failure must be caused by a chance unfortunate placing of the sulfosin depôt. Each series of treatment has included 8 injections, and a new injection has never been given until the effect of the former has absolutely passed, this lasting 3-4 days. Often there has been a certain parallelism between the amount of sulfosin injected and the rise in temperature, but this was far from always the case, which may again have been due to the placing of the depôts. A certain habituation also takes place, but this only applies to each single series; on starting a new series small doses must again be used (Harris).

Regarding the biological effect of sulphur, I shall refer to Schroeder's[1, 2, 3] reports; it must here be mentioned that the most conspicuous effect, and the effect most easily controlled, is the characteristic rise in temperature, which appears quickly during

Temperature after injection of sulfosin (↓).

the first 12 hours, the temperature again falling rapidly for the next 12 hours; then there is again a small rise, which is characteristic of the sulfosin temperature and which, *e.g.*, is not seen in

the malaria temperature, the former therefore being of longer duration than the latter; according to Schroeder[2] and others the rise in temperature is due to the formation of complex sulphur compounds—sulphur-albuminates—produced by the destruction of muscular tissue, and must be looked upon as indicating that the defence of the organism has produced substances causing the rise in temperature, perhaps through a heat regulating mechanism. It is a great advantage that by dosing the sulphur, it is possible to regulate the height of the temperature.

During the treatment there may be loss in weight, which is generally quickly regained, but there may also be an increase in weight. The sedimentation rate of the red blood cells increases —possibly as a result of the rise in temperature, and the leucocyte count always increases. Often there is a slight fall in haemoglobin percentage and in red cell count (destruction), followed by a rise—often to values above the original ones. The blood pressure may also fall slightly during the rise in temperature, but never dangerously as has been observed in malaria treatment. The cardiac and respiratory rates may increase slightly. The Wassermann reaction in the blood and spinal fluid may become negative, and the cytological changes in the spinal fluid may become normal during the treatment (Schroeder).

Of course, the sulfosin injections may be followed by certain inconveniences; chills, or more seldom, cold shivers. There may be headache, nausea, and rarely vomiting; there may also be pain at the site of the injection, lasting whilst the temperature is increased. Sometimes there has been infiltration at the site of injection, and in some very rare cases, formation of abscesses.

According to Schroeder albuminuria or other symptoms from the kidneys or heart have never been met with; neither have jaundice nor symptoms of intoxication been observed, but a focal reaction with inflammation of the antrum of Highmore has been seen, and Loberg thinks that he has seen a case of old tuberculosis break out again after treatment with sulfosin; these unfortunate effects are, however, quite negligible compared with the enormous number of injections that have been given, and all in all this remedy may surely be called harmless.

Harmful effects on the eyes have never been observed, whilst as already mentioned, malaria treatment may be accompanied by complications on the part of the eyes.

The processes which have first of all been considered to play a part in treatment with sulfosin are as follows:—(1) the rise in temperature, (2) leucocytosis, (3) increased metabolism with removal of harmful substances, (4) vaso-dilatation, (5) counter-poisoning and possible production of antibodies.

The rise in temperature is most likely of great importance, but

whether the effect is to be understood as merely mechanical, the temperature simply destroying the harmful agent, or whether more complicated processes take place, is not known. As far as treatment with malaria is concerned, Clark maintained that there is an effect on atrophy of the optic nerve even though the temperature does not rise, but this point of view is fairly unique, and—as will be demonstrated later—it hardly applies to treatment with sulfosin. Leucocytosis is definite, but it is hardly alone responsible for the effect (vide also Power). The increased metabolism is partly connected with the rise in temperature, and neither the increased metabolism alone is able to explain the effect, *e.g.*, administration of thyroid is not able to influence atrophy of the optic nerve. Vaso-dilatation undoubtedly takes place ("new" vessels on the optic nerve after injection of sulfosin in cases of atrophy), and probably it plays a certain part. It is not known whether any special antibodies are formed. The effect of sulfosin must most likely be understood in the following manner:—The rise in temperature causes a counterpoisoning, removal of the harmful agent; simultaneously the blood vessels dilate and cause more ample transudation in the surrounding tissues, which together with the increased metabolism of the cells removes the harmful agent, of whatever kind it is.

The patients to be dealt with here have on the whole tolerated the treatment with sulfosin well; in two cases the treatment had to be discontinued—once in a patient with atrophy of the optic nerve on account of vomiting and once in a patient with keratitis. In some few cases cold shivers were seen during the rise in temperature, and attacks of perspiration when the temperature fell; but otherwise tenderness and pain at the site of the injection (appearing 3-4 hours after the injection had been given and lasting as long as the temperature was raised), headache, malaise, and in a single case nausea, have been the greatest discomforts; many patients have not felt any ill-effects at all from the treatment. In one case (473/35) a hypopyon was observed during treatment, but it disappeared when treatment was continued (it may possibly have been present before the treatment was commenced, but not have been visible on account of the opacity of the cornea, and first became visible when the opacity had cleared). In one instance there was a large infiltration at the site of injection (56/34); it disappeared quickly after hot fomentations had been applied, and in one case (487/37) a large abscess, which had to be incised, developed after the 11th injection. One patient (387/37) had a scaly tuberculous syphilide on his upper lip; this syphilide decreased considerably during treatment with sulfosin (*cf.* Schroeder[4].)

Since 1933 17 cases of parenchymatous keratitis have been

treated, and in order to evaluate the results, they have been compared with the cases of parenchymatous keratitis during the period 1930-1937 which have not been treated with sulfosin, 22 in all.

Both groups include adults as well as children, and several of these cases have verified the fact that anti-syphilitic treatment unfortunately does not protect against parenchymatous keratitis. One case—amongst the sulfosin treated ones—was of parenchymatous keratitis in the second generation (82/36). Not all cases of parenchymatous keratitis since 1933 have been treated with sulfosin, the cases having been selected in disfavour of sulfosin treatment, all patients on admission besides local treatment (atropine, fomentations, perhaps cocaine or scopolamine) received the general treatment used so far in the department: diaphoresis with acetyl-salicylic acid, infusion of sarsaparilla or hot lemon juice, syrup of iodine and mercury, ordinary anti-syphilitic treatment with bismuth and neoarsphenamin, and during the later course of the disease: dionin, Pagenstecher's ointment, arc-light, and in a few instances (before 1933) subconjunctival saline; the cases which improved in this manner were not given sulfosin.* In some cases, therefore, a fairly long period of time elapsed between the onset of the disease and the commencement of treatment with sulfosin, in cases 56/34 and 378/34 about 3 months; in such cases I have when judging the effect of sulfosin only reckoned from the time treatment with sulfosin was commenced, which seems justifiable, as in these cases the course of the disease perfectly resembled that of the cases which were treated with sulfosin shortly after their admission, which were 11; the former group stayed 56·7 days on average at the hospital, the latter group —the cases treated at once—47·2 days. The local treatment was of course continued together with the sulfosin treatment. In 11 cases ordinary anti-syphilitic treatment was commenced before or simultaneously with the sulfosin, and it was strange to see that if lacrimation, photophobia and injection had disappeared by the help of sulfosin, these phenomena might reappear when bismuth (487/37) or particularly neoarsphenamin were given (587/33, 56/34, 372/36); if, on the other hand, the irritation phenomena had subsided some time before this general anti-syphilitic treatment, they did not reappear.

As already mentioned, sulfosin has an excellent anti-syphilitic effect, the Wassermann reaction improved in the blood and spinal

* That the sulfosin-treated cases are the most severe may also be seen from the fact that 18 out of 29 affected eyes had precipitations or membranes, whilst only 15 out of 37 cases not treated with sulfosin showed these complications.

† The longer stay in hospital of the cases which were treated late with sulfosin may perhaps also be explained by the affection in these cases having spread to a larger part of the cornea, and the cornea in these cases mostly being amply vascularized.

fluid, and cytological changes in the spinal fluid become normal, which was demonstrated by Schroeder, which I have sometimes had the opportunity of observing, and therefore there does not seem to be any danger in postponing anti-syphilitic treatment till the irritation of the eyes has subsided.The phenomenon that bismuth and neoarsphenamin cause reappearance of the irritation processes must most likely be looked upon as a sort of Herxheimer reaction.

Amongst the group not treated with sulfosin, 18 had positive Wassermann reaction, the other four had previously been treated with anti-syphilitic agents. Amongst the sulfosin treated cases, 15 had positive Wassermann reaction, 2 negative (previously received anti-syphilitic treatment).

The most conspicuous feature during treatment with sulfosin is the fact that the process in the eyes so quickly calms down; in most cases it has been remarked in the records often after only one injection (*e.g.*, 522/34, 198/36, 372/36), or after a few injections (*e.g.*, 365/36), that the eyes have grown paler or white, that they open more easily, that photophobia and lacrymation have decreased—all signs that the irritability has been reduced or has disappeared, and very often these very painful symptoms have subsided after a few days, much quicker than previously during diaphoresis, when the eyes might indeed during or after treatment become more irritated, a healed parenchymatous keratitis might even reappear and the Wassermann reaction become more posititve (408/31).

I have also found another indication of the quickening effect of sulfosin upon the healing of the eyes in the fact that a pupil which did not respond to large doses of atropine or cocaine became dilated after one or a few injections of sulfosin and could now be kept dilated with comparatively small doses of atropine, the eye at the same time growing paler and the injection of the iris, always present in such case, subsiding. A similar effect has only been reported during treatment with malaria (Meller). The pallor following sulfosin seems reliable; recurrence of the redness has at any rate not been observed, such as was quite often the case during the treatment previously used at our department, and such as Schreiber has described following treatment with malaria.

In order to show in figures that the hospitalisation period becomes shorter and the irritation phenomena disappear more quickly during treatment with sulfosin, I have put down in a table: (1) The average number of days the patients have stayed in hospital on account of their parenchymatous keratitis (if the stay has been particularly long on account of light treatment. etc., or complications not directly concerning the keratitis, the period has only been calculated until the eye-affection was healed):

(2) The average number of days which passed until it was put down in the record that the eye was white or nearly so.

The results are:

(1) In the cases of parenchymatous keratitis in the first eye the stay in hospital was on average of the same length in the cases treated with sulfosin as those not treated.

In cases of parenchymatous keratitis in the second eye, this period was considerably shorter (about half) when the first eye had also been treated with sulfosin (only one case that was treated with sulfosin for the second eye had not received sulfosin for the first eye).

If both eyes are attacked simultaneously, there has been a moderate shortening of the stay at hospital as compared with the cases not treated with sulfosin.

(2) In the cases treated with sulfosin, the average number of days that have passed till it has been remarked in the record that the eye had become white or nearly so were not quite 2/3 the number of days that passed in the cases not treated with sulfosin when the first eye was attacked, about 1/3 when the second eye was attacked, and 1/5 in the cases where both eyes were attacked. It may, therefore, be said to be a general rule that: *both the length of the stay at hospital as well as the time that passed until the eyes became white* (which means that all irritation, photophobia, lacrymation, etc., has disappeared) *was considerably shorter in the cases treated with sulfosin than in those not treated with sulfosin,* and even then it must be kept in mind that the sulfosin treated cases were the most severe.

The dimness of the cornea may clear astonishingly quickly during sulfosin treatment, *e.g.*, in case 82/36, in which the acuity of vision in 16 days increased from being able to count fingers at 1 metre's distance to 5/12-5/18, even though there were some precipitates, and—as also in some other cases—only a few deep vessels could be seen in the cornea; these deep vessels have been considered of great importance to the clearing. How the dimness clears when there are only a few vessels is difficult to understand; it is possible—as suggested by Schroeder and others—that following the injection sulfosin is converted into polysulphides which circulate in the organism and when meeting inflammatory or degenerative processes, they increase the metabolism of the cells; in this special case they must then increase the stream of fluid in the cornea, thereby removing the inflamed tissue.

The fact that the dimness may disappear quickly and with only little or no assistance from deep vessels disturbs the dogma that vascularisation is of fundamental importance to the absorption of corneal dimness, and at any rate to a certain degree upsets the usual sharp division of parenchymatous keratitis into three

stages, used in Scandinavian and German publications. Sometimes the vessels themselves or remains of them may be the cause of slow clearing, for often the cases that clear slowly contain many vessels; and the records show that the cases that had few vessels prior to sulfosin treatment cleared more quickly than the cases with many vessels, *viz.*, sulfosin should be given early, before there are many vessels.

The table (p. 612) moreover seems to demonstrate that when bismuth or bismuth together with neoarsphenamine were given immediately before or simultaneously with sulfosin (13 out of 19 eyes) many vessels appeared in the cornea and healing was slow (after 4 months only two had obtained an acuity of vision > 6/18), whilst the eyes (10) which were first treated with sulfosin and later with anti-syphilitic remedies only presented deep vessels four times (and these vessels were present before treatment with sulfosin was commenced), and before the end of 4 months the acuity of vision had become > 6/18 in 6 cases. It must be added that both groups include cases that healed very slowly, but on the whole the cornea cleared more slowly in the cases that were not treated with sulfosin, even though they presented many vessels. There are, however, also some cases with few vessels amongst those that first received anti-syphilitic treatment and later sulfosin.

In per cent. the number of cases which after 4 months had obtained an acuity of vision > 6/18 are about equal amongst the sulfosin treated cases and the non-sulfosin treated ones (about 40 per cent.). But it must here be kept in mind that the sulfosin treated cases were the most severe—those that had not responded to previous treatment and those with the greatest number of complications.

In 15 out of 37 eyes not treated with sulfosin there were precipitates to some extent, and in 18 out of 29 sulfosin treated eyes, but only in two of the latter cases did the precipitates appear after treatment had commenced, in the other cases they were present beforehand.

One of the most interesting questions is: is sulfosin able to prevent the development of parenchymatous keratitis in the other eye? According to Dalsgaard-Nielsen parenchymatous keratitis appears on both sides in 83 per cent. of the cases (time of observation 1-56 years), and generally the two eyes are attacked at intervals of days or weeks (Schieck), more seldom months or years pass; most often the disease is equally severe in both eyes, the duration and intensity being the same, but the deviations from this rule are so numerous, that it is not possible to predict anything regarding the second eye; this is also in accordance with the figures of my non-sulfosin treated cases (first eye: on average 49·1 days, second eye 51·2 days). As already mentioned the attack on the

	Duration of stay in hospital (days)			Days before eyes became white or nearly so (days)			M.	W.	C.	Total	Bilateral k.p.			In 4 months > 6/18	Vessels in cornea		+ Precipitates + + Membranes
	1st eye	2nd eye	Both eyes	1st eye	2nd eye	Both eyes									many	few or +	
+Sulfosin	49·1	51·2	74·5	51·6	52·6	159·5	...	...	...	...	...	...	...	...	...	...	...
No. of cases	9	9[2]	4	9	10[4]	2	4	9	9	22[5]	15 = 68%	No. of eyes Vision determined	(37) 29	12= 41·5%	29[8]	6	16
+Sulfosin	50·8	28	65·3	39·1	15·8	34·4	...	...	...	...	...	...	...	...	...	...	...
No. of cases	9	4	7[3]	9	4	8	3	5	9	17	4 = [6] 44·5%[7]	No. of eyes Vision determined	(29) 20	8= 40·0%	17[11]	12	18[10]
Antisyph. treatm. together with, or before sulfosin	60·8	(26)	67·3	39·8	(10)	31·7	...	...	...	...	...	No. of eyes Vision determined	(19) 12	2	13	6	13
No. of cases	4	1	6[3]	4	1	7	2	4	5	11	1[6]	...	...	...	...	...	...
Sulfosin alone and later antisyph. treatm.	42·8	28·7	(53)	38·6	17·7	(53)	...	...	...	...	...	No. of eyes Vision determined	(10) 10	6	4[9]	6	5
No. of cases	5	3	1	5	3	1	1	1	4	6	3[6]	...	...	...	...	...	...

[1] Not all records can be used for calculating useful figures. For each group "No. of cases" indicates how many cases have been used for calculating the average.

[2] Six cases had previously had k.p in one eye.

[3] Duration of stay in hosp. not known in one case.

[4] *Vide* case 189/31.

[5] Three cases were moved to other departments and cannot be used for calculating the duration of the disease, but for calculation of the bilateral cases.

[6] The number indicates k.p in 2nd eye *after* S. in 1st eye.

[7] Percentage calculated from No. of cases of keratitis in 1st eye (only 23·5 per cent. of total 17).

[8] With two eyes vessels not mentioned.

[9] In all cases many vessels before S.

[10] In 16 eyes before S. In two eyes after S., in both cases A.T. anticipated S.T.

[11] In 15 eyes before S.

second eye was shorter and milder (first eye 50·8 days, second eye 28 days) if the attack on the first eye had been treated with sulfosin than if it had not, and this already answers our question; no more than any other treatment is sulfosin able to prevent the development of parenchymatous keratitis in the second eye, but amongst 22 non-sulfosin treated cases 16 were bilateral (68 per cent.—time of observation 15 months to 8 years) whilst among 9 cases treated with sulfosin in the first eye, only 4 developed keratitis in the second (44·5 per cent.—time of observation 6 months to 5 years). When the keratitis develops in the second eye very soon after the first eye (days to weeks) preventive effects of sulfosin cannot be expected, precursors of the keratitis already having developed in the cornea before treatment was commenced. Closer examination, however, reveals that in 4 cases of parenchymatous keratitis in the first eye, in which anti-syphilitic treatment was administered immediately before or simultaneously with sulfosin, there was only one attack in the second eye, which agrees well with Carvill, Coulter and Ingram's and K. Langendorff's opinion that anti-syphilitic treatment may act as a prophylactic against keratitis in the second eye; in the five cases that were treated with sulfosin first and later with anti-syphilitic treatment, parenchymatous keratitis developed in the other eye in three cases. These figures are too small to make any rule, but it seems that though sulfosin alone—followed by anti-syphilitic treatment—gives a shorter period of disease and quicker healing, sulfosin simultaneously with anti-syphilitic treatment is better able to protect the other eye. The result of this seems to be, that it must be best to start with sulfosin treatment, and as soon as the eye has reached a quiet stage, to commence with intense anti-syphilitic treatment. This should give a chance of avoiding parenchymatous keratitis in the other eye.

In both groups—the sulfosin treated cases and the non-sulfosin treated ones—the affection healed more quickly in children than in adults.

It has not been possible to demonstrate any effect of sulfosin upon other manifestations of congenital syphilis.

Syphilitic Atrophy of the Optic Nerve treated with Sulfosin

The usual anti-syphilitic treatment with arsphenamine, bismuth or mercury has not been very successful against syphilitic atrophy of the optic nerve; in spite of intense treatment—Weinberger even says: *because* of it—the atrophy of the optic nerve proceeds, and the defence for continuing the use of anti-syphilitic treatment has been, that a beneficial effect is supposed to have been seen upon the other tabetic symptoms, and further that the presence of basal

meningitis can never be excluded with certainty. A review of the treatment that has been used shows how much uncertainty has prevailed (pills of silver nitrate, electricity, injections of strychnine, endo-lumbar injections of arsphenamine, injections of gas or arsphenamine into the cerebral ventricles, retrobulbar injections of atropine, opening of the ethmoidal cells or the sphenoidal sinus, fever, etc.), none of which has shown any results of importance. When Wagner v. Jauregg's malaria treatment of tabes appeared, much was anticipated in the way of treatment of atrophy of the optic nerve, as this treatment had such an excellent effect upon other tabetic symptoms, and tabes and atrophy of the optic nerve are systemic diseases—often closely related—one being localised to the posterior columns, the other to the optic nerve. Malaria treatment of atrophy of the optic nerve both has adherents (Wagner v. Jauregg, Elschnig, Hessberg, Bruno Fleischer, Clark, and others), who have seen improvement following this treatment, and opponents (Bæhr, Jænsch, and others) who have on the whole taken up a very nihilistic attitude towards treatment of atrophy of the optic nerve, who also from theoretical reasons are of opinion that malaria treatment of atrophy of the optic nerve is not beneficial.

One of the reasons for this disagreement between the different authors seems to be, that they have not made it clear what one may expect from treatment with malaria. Those who expected a conspicuous improvement were of course disappointed, a fever treatment cannot work miracles, and the nerve fibres that have already been destroyed before treatment was commenced cannot be made to function again. Slight improvement is of course possible, as fibres which were not quite destroyed, only "paralysed," may resume their function—partially or totally—if the harmful agent, of whatever kind it is, can be removed or neutralised during treatment; it is a process of this kind which I have imagined to be the true cause of the improvements observed both during treatment with malaria and with sulfosin; but on the whole an arrest of the process is the most favourable effect to be expected. If this is not achieved, a slower progress of the disease after the treatment than before is to be hoped for, but this may be very difficult or quite impossible to judge. Lastly it must be kept in mind, that if the disease is not arrested by the treatment, the latter should not be blamed, but it should be remembered that without treatment or treated according to previous methods the disease invariably proceeds more or less quickly.

Treatment with malaria may, however, give reason for certain scruples, and where atrophy of the optic nerve is concerned they are not quite the same as in parenchymatous keratitis. The fever of malaria may only to a certain degree be regulated with quinine

—and quinine is not a good remedy for an already damaged optic nerve (quinine-amblyopia)—which gives rise to some contra-indications: advanced age, weakness, diseases of the heart and vessels, kidney disease; and lastly treatment with malaria can only be used in closed departments, as malaria strains must always be available, whilst—at any rate in this country—the risk of infection does not count. This is probably the reason why malaria treatment of atrophy of the optic nerve has not been taken up in this country; but of course, if this treatment is superior to treatment with sulfosin, it must decidedly be preferred in so serious an affection, which according to Wilbrand and Sänger attacks 13-15 per cent. of all tabetics and 8-10 per cent. of the patients with general paresis, and affects relatively young individuals of 30-40 years of age, and in 2-3 months to 12 years leads to blindness without remissions of any importance (on average in 2-3 years).

The available literature seems to indicate that there is not any great difference between the results of treatment with malaria and with sulfosin (vide *e.g.*, Wagner v. Jauregg: slight cases: sometimes standstill and sometimes improvement; severe cases: proceed, but more slowly. Bruno Fleischer, Weskamp, Tereskeli), but these publications suffer from the fault that the time of observation has been too short. Perhaps malaria treatment also involves a greater risk of complications; Clark has observed neuro-retinitis (bismuth ?).

Fifty-four cases of atrophy of the optic nerve treated with sulfosin have been published. Sixteen improved, 15 did not change, 7 became worse, during a period of observation of 6

Cases of atrophy of the optic nerve treated with Sulfosin

AUTHOR	Better	Unchanged	Worse	Total	
Canella - - 1931	...	4	....	4	
Fried (Winkler) - 1931	6	4	2	12	
Kallmann - - 1932	...	2	...	2	
Smith - - 1932	1	...	...	1	
Tirelli - - 1932	3	1	1	5	
Weinberg - - 1932	...	...	...	...	16 results not stated
Busacca - - 1933	...	3	1	4	
Larsen - - 1933	3	1	3	7	
Schroeder - - 1933	1	...	...	1	
Winkler - - 1935	2	...	...	2	
Total - -	16	15	7	38	+16=54

months to 4 years; the result was not stated in 16 cases. Cases in which it was certain sulfosin had reduced the vision have not been reported.

During the years 1932-1938 14 cases in all of syphilitic atrophy of the optic nerve have been treated at the eye department of the Municipal Hospital. The age of the patients was between 31 and 61 years at the time when the disease broke out, and the time of infection with syphilis, when known, was 12 to 28 years previous to the time when the eye disease caused the patient to consult a doctor. The Wassermann reaction was positive in the blood in all cases, and in the spinal fluid in 9 cases (very strongly so in 3 cases) (in 2 cases spinal puncture was not carried out). The number of cells and the values for albumen were raised in most cases, the globulin values being normal. Three patients did not know anything of their infection, and 2 patients did not know what treatment they had received. The remaining 9 patients had been treated with varying intensity, most often not very well, which is again connected with the fact that they had not put in appearance for control. There does not seem to be any difference between the course of the sulfosin treated cases of atrophy of the optic nerve in the cases treated well with anti-syphilitics and in those badly treated; the cases that had not been treated at all seem to show a tendency towards bad results. One patient (No. III) had, however, been treated very well with anti-syphilitics, but the result of sulfosin was extremely poor; how great a part sulfosin has played in this is, however, difficult to say, as already before treatment was commenced vision was rapidly failing (during the month he waited for admission his acuity of vision fell from 5/24 to 5/36 and 5/60, and the field of vision for red, which to begin with was only slightly reduced, was quite lost; further he had severe myopia: right -5·5, left -10) and it cannot be excluded that these facts have contributed towards the primary bad result (appearing in immediate connection with the treatment). All the patients had other tabetic symptoms—more or less pronounced—unequal pupils not responding to light, areflexia, darting (lancinating) pains, pain on pinching at heels.

Ophthalmoscopic examination revealed atrophy of the disc, in most cases diffuse. The result of sulfosin treatment on these symptoms has previously been thoroughly investigated (amongst others by Schroeder). Most often 3-4 injections of bismuth were given before treatment with sulfosin was commenced, later arsphenamine was generally administered. The present data do not reveal any difference between the course of the disease in the cases treated with sulfosin alone and those also given bismuth; neither do they reveal any difference between the patients with weak Wassermann reactions in blood and spinal fluid and those

with strong sero-reactions, though there seems to be a tendency towards a better effect of sulfosin in the cases with weak reactions; it should, however, be emphasized that none of my cases shows any parallelism between the reactions in the blood and in the spinal fluid, the differences may even be large: *e.g.*, cases I and VII.

It has already been mentioned that sulfosin alone may improve the sero reactions, but whether improvement will continue for three years, as observed by Wagner v. Jauregg,[2] following treatment with malaria, cannot be determined from my investigations, if not for other reasons because none of my patients received sulfosin alone for such a long period of time.

Treatment with sulfosin was carried through in 12 of the 14 cases; they all showed a good temperature reaction. In case IV treatment had to be discontinued on account of nausea and vomiting and the patient not wishing to continue; in case XI 6 injections were given and the temperature rose well, but the patient had aortitis; she got palpitations and pronounced cold shivers, but even though this need not indicate any danger (*cf.* Schroeder) the treatment was discontinued. After the first injection the rise in temperature was always moderate—38°-38·5° C.—as recommended by Schroeder and Dreyfus and Mayer, and with later injections a greater rise was aimed at. As in the patients with parenchymatous keratitis, the discomfort following the injections was but slight, and also as in the patients with parenchymatous keratitis, sulfosin was administered in a series of 8 injections. 6 patients received more than one series. In 3 patients (II, III, VII), two series were given at once or at short intervals (case VII was moved to another hospital and received 19 injections in all). These three patients showed a tendency towards getting worse already during treatment. Three patients were given a new series 2-4 years after the first, at a time when their vision had again begun to diminish; these cases, which had been beneficially influenced by the first series, were also beneficially influenced by the second (cases I, IX, XII). From these facts it is perhaps possible to draw the conclusion that if the first series does not cause a standstill or perhaps slight improvement of the affection, it is not worth continuing the treatment, the disease in these cases not being affected by sulfosin in spite of a good fever reaction; on the other hand, it seems that if the first series has been of benefit, it is reasonable to give another or several more series later, if vision should again be liable to decrease, in which otherwise desperate cases there should be some hope of preserving what vision there is or preventing blindness. This again means that the patients must be controlled fairly often and must be told to come again immediately for repeated treatment with sulfosin, if their vision should again decrease.

Several patients observed that during the first days after an injection their acuity of vision and field of vision diminished, only to become clearer again (*e.g.*, case XII, whose vision on admission was 3/36, during treatment he could only count fingers at a distance of 2 metres, and when discharged his vision was 6/60). Ophthalmoscopic examination some hours after an injection has sometimes revealed that the disc no longer has the same white, porcelain colour, but is more grayish, and new vessels were observed on the disc (also observed by Fried). Sometimes the limits of the disc were blurred, but there was no definite prominence. Neither the change in colour, nor the vessels, however, were permanent, even though the treatment caused improvement. It is not easy to explain these variations in the acuity of vision during treatment, perhaps treatment with sulfosin gives rise to a greater blood flow and richer diapedesis through the walls of the capillaries, which may again—purely mechanically, by pressure—or through chemical influence (or both) affect the optic nerve, its function temporarily decreasing; when the inflammatory products have been removed and the swelling caused by the new vessels and the diapedesis has again subsided, the function is restored. The new vessels, the swelling of the discs with blurred limits and the fact that most often central vision and not the field of vision was affected (the central fibres are the most fragile and easily damaged) together with the fact that parenchymatous keratitis clears without any co-operation of vessels of importance, as well as Ipsen's determinations of temperature, might indicate that this opinion is correct. If this is the case, we cannot deny that there is a theoretical risk of the pressure permanently damaging the optic nerve (this, however, does not seem to have been observed).

There are, however, also cases in which vision improves gradually during the whole treatment, *e.g.*, case VIII, in which the

TABLE I

	ACUITY OF VISION	FIELD OF VISION
Improved	VIII X XII XIII XIV	I V VII VIII IX X XII XIII XIV
Unchanged	I V VI IX XI	II VI XI
Worse	II III VII	III

Cases on first admission compared with condition at discharge.

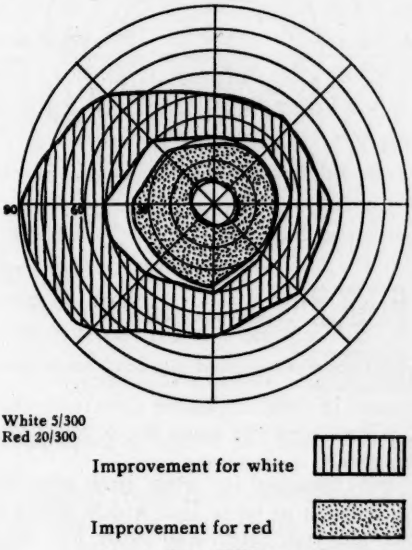
TABLE II

	ACUITY OF VISION	FIELD OF VISION
Improved	VI VIII X	I VI VIII X XII XIII
Unchanged	I XII	VII
Worse	II III VII IX XIII	II III IX

Cases on first admission compared with condition at present moment.

vision improved upon one eye from < 6/36 with no colour distinction to 6/18 and distinction of 10/300, and case X, in which vision improved from 6/12 to 6/9 without any reduced interval.

Table I shows the immediate results of the treatment, the conditions on admission and at discharge being compared. In 5 cases the acuity of vision had improved, in 5 cases it was the same; but the field of vision had increased in 9 cases and had not changed in 3. Only in one case (III) both acuity and field of vision decreased. The improvements are not quite small; thus in case XII the acuity of vision improved from counting fingers at a distance of 1 metre to 3/36, and case XIII improved in field of vision as seen on p. 601. It is characteristic that the field of vision generally showed the best and most prolonged improvement, which probably is due to the central fibres being more fragile and more easily damaged than the peripheral ones, as is often seen also when the optic nerve is damaged by pressure, in which case a central scotoma may be the only impairment in the field of vision. It is also remarkable that the visual field for colour improves, or reappears in cases where it has been lost. Case VIII of his own accord said that some time after he had been discharged he was able to see the colours of the flowers in his garden, and case II said that on his discharge he was able to tell the colour of the traffic lights.



CASE XIII.

Vision right eye, no perception of light. Vision left eye, 5/18 + 0·5.

The improvement in central vision is small (except in case VI, which is not quite comparable, as the refraction was not determined at the first examination).

Improvement or standstill of the affection may last, as seen in Table II, in which acuity of vision and field of vision have been compared from the time of the first (and in some cases only) stay in hospital with the present time. It is seen that in three cases central vision improved, and in two cases it was the same; in the same 5 cases plus one more (6 in all) the field of vision had also increased. In 3 cases both acuity and field of vision had deteriorated; in 2 cases acuity of vision deteriorated, whilst in one of these cases the field of vision increased and in the other it did not change. It seems to me that these results are relatively good, and the period of observation has been fairly long, as seen in table VI; the improved cases were observed for 5 and 6 years respectively; but the table also shows that some of the most recent cases have given bad results, which most likely indicates that it is not possible to influence the disease in these patients with sulfosin, either because the disease is too severe, or because the patients in themselves are refractory to sulfosin; but on the other hand, it would hardly be just to say that sulfosin has aggravated the affection, which must simply be said to have progressed. 4 cases have not been put down, as they did not turn up for control examination.

TABLE III

	ACUITY OF VISION	FIELD OF VISION
Improved	I VI IX	I II V VI VI I IX X
Unchanged	II III V VII VIII X XI	VII XI XII XIII
Worse	XII XIII XIV	III XIV

Cases at discharge compared with condition 1 year later.

TABLE IV

	ACUITY OF VISION	FIELD OF VISION
Improved	VI	VI
Unchanged	VIII X XII XIII	I VIII X XII XIII
Worse	I II III VII IX	II III VII IX

Cases 1 year after discharge compared with condition at present moment.

The improvement in field of vision generally lasts the longer, *e.g.*, in case X it has been the same for 4 years after the patient had been discharged.

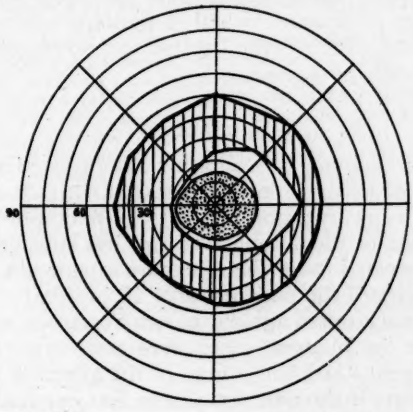
Improvement may proceed for some time after the patient has been discharged, as seen in table III, which shows the conditions at discharge and one year later, and table IV, which shows the present condition compared with the condition one year after the

LENGTH OF OBSERVATION

CASES	Years	Improved	Unchanged	Worse	Unknown	Total
I II III IV ...	6	1	—	2	1	4
V VI VII ...	5	1	—	1	1	3
VIII IX X ...	4	2	—	1	—	3
XI XII	3	1	—	—	1	2
XIII XIV ...	2	—	1	—	1	2
TOTAL	—	5	1	4	4	14

Cases are called improved, when either acuity or field of vision (or both) are improved and the other unchanged; unchanged when acuity or field of vision is improved and the other worse (or both unchanged).

The "unknown" did not answer questionnaire April, 1938.



CASE X.

Vision right eye, perception of light (no projection). Vision left eye, 5/12.
(Third admission)

patients had left hospital; these cases show that the improved cases by and by become stationary, but unfortunately, other cases have been aggravated. The fact that improvement may proceed for some time after treatment has been concluded, seems to indicate

that the counterpoisoning and removal of the harmful agent goes on for some time after the cure is ended, and that it takes some time for the nerves to begin their function again; the tables further show that on the whole the cases which grow worse are those which during the whole course of treatment have had a tendency towards aggravation, even though the process periodically may stand still, as in cases II, III, VII. Naturally enough the cases which are most accessible to treatment are those which proceed slowly. Difference between the effect on recent and on old cases is not demonstrable. The best results seem to have been gained about one year after treatment.

Strangely enough cases XI and XII do not show any signs of affection of the normal eye, in spite of a period of observation of 3 years, and otherwise one-sided syphilitic atrophy of the optic nerve is indeed rare—usually both eyes are affected at intervals of months—more seldom years (Willbrand and Sänger); but it is not possible to determine how great a part sulfosin has played in this phenomenon. These cases must be watched with special interest in order to examine whether perhaps sulfosin treatment of syphilitic optic atrophy may be able to prevent affection of the second eye in unilateral cases.

Summary

17 cases of parenchymatous keratitis were treated with sulfosin, and the results of this treatment compared with 22 cases that did not receive sulfosin; irritation of the eye—photophobia, lacrymation and injection of the conjunctiva and iris subsided far quicker in the sulfosin treated cases than in the non-sulfosin treated ones, which also shortened the patients' stay in hospital.

Sulfosin did not protect against parenchymatous keratitis in the second eye, but the bilateral cases were more rare (49·5 per cent. against 68 per cent.) and the course of the affection of the second eye was less severe if the affection of the first eye had been treated with sulfosin, than if it had not been so.

Most often only few vessels were seen in the cornea following sulfosin treatment, but these cases cleared just as quickly as the non-sulfosin treated ones with many vessels, in spite of the sulfosin treated cases being more severe.

14 cases of tabetic atrophy of the optic nerve observed for 2-6 years are described. It seems without doubt that treatment with sulfosin is able—at any rate temporarily—to check the atrophy of the optic nerve (in certain cases it may cause some improvement); but some cases seem inaccessible to this treatment and progress. Both the central vision and particularly the field of vision and colour vision may improve, and this improvement may continue for as much as one year after the course of treatment has been ended.

5 cases in all improved, 1 case did not change and 4 cases were aggravated, whilst the further destiny of 4 cases is unknown.

If a patient who has previously been treated with sulfosin again shows signs of declining vision, a new course of treatment should be given, as the effect will resemble exactly that of the first course.

Two cases of unilateral atrophy do not show any signs of affection of the normal eye in spite of a period of observation of 3 years.

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TABLE V.—CASES OF ATROPHY OF THE

Case No.	Record. Sex. Age	Years since Syphilis Infection.	Treatment.	Symptoms.	Acuity of Vision.	Condition of Eyes.	Wassermann Reaction (blood) Kahn.
I	372/32 Male 49	25	1 series of mercury inunctions	For 2 years declining vision, cannot read. +Paraesthesias in feet and pain at back of thighs. Voids urine incontinently. Difficulty with test phrases. ÷K.J. ÷A.J. -P.H.	R. eye counts fingers 3 mm. L. eye ibid. Colour vision extinct both eyes	+Argyll Robertson syndrome. Discs sharply outlined porcelain white, diffuse atrophy	0·60100 0 40
II	503/32 Male 31	12	2 series of mercury inunctions	For 6 months declining vision especially for reading. K.J. ÷A.J. -P.H.	R. eye 5/60 L. eye 5/36 No colour distinction	+Argyll Robertson syndrome R. pupil > L. irregular Discs sharply outlined, white diffuse atrophy	20100 0 20
III	432/32 Male 43	18	Neo-arsphenamin. ab. 300 mercury inunctions	For 2 months declining vision rapidly, mostly right eye. Red vision lost in one month. +K.J. -A.J. Difficulty with testing phrases.	R. eye 5/60-55 L. eye 5/36÷10·0 Colour vision extinct both eyes	Pupils respond normal Discs diffusely pale +Myopic cornea	20100 0 40
IV	387/32 Female 56	16	Mercury inunctions	For 1 year declining vision. For 6 years darting pains in legs. +Paraesthesias and dead-feeling in arms and legs. -K.J. -A.J. +Ataxia +Romberg	R. eye 2/36+2·0 L. eye counts fingers in front of eye Colour vision extinct both eyes	R. pupil > L. +Argyll Robertson syndrome Discs sharply outlined Diffuse atrophy	000401 100 0 320
V	JC/1932 Male 47	15	Inspiration of mercury gas	For 6 months declining vision. No other symptoms.	R. eye 6/18÷1·0 L. eye 6/12 Colour vision extinct both eyes	Pupils irregular, slight response to light, no accommodation response, slight temp. atrophy both eyes	20100 60 20
VI	HH/ 1932 Male 47	20	÷ treatment since 1930	For 2 years declining vision. No other symptoms. Syphilis discovered 1930 when vision began to decline.	R. eye counts fingers 1m. L. eye no sense of light No sense of colour both eyes	R. pupil > L. Slightly irregular no response to light nor accommodation, sharply outlined discs, diffuse atrophy	0·40100

OPTIC NERVE TREATED WITH SULFOSIN.

Case No.	Spinal Fluid	No. of S. Injections Maximum Temp. Auxiliary Treatment.	Discomforts.	Acuity of Vision at end of Treatment.	Acuity of Vision 1 year later.	Acuity of Vision at present moment	Field of Vision.	Total Result. Remarks.
I	000601 00 0 160 Cells 228/3 A 30 G 1	8 S. inj. 40° 12×1.5 cc. bism. 1935 8 S.inj. 39.6°	Pain at back of thighs	R. eye counts fingers $2\frac{1}{2}$ m. L. eye counts fingers 2 m. No colour vision, field of vision the same	R. eye 4/36 L. eye 2/36 Field of vision increased	R. eye counts fingers 3 m. L. eye movements of hand in front of eye	As 5 years ago	Central vision the same. Field of vision improved. The vision, which in 1935 had begun to decline improved after a second series of S.
II	— 10 Cells 310/3 A 18 20 G.1	8 S. inj. 39° 12×1·5 cc. bism. 2 months later again 8 S. 39°	—	R. eye counts fingers 3 m. L. eye 5/36 No colour vision, field of vision slightly reduced	Ibid. Ibid. Sees red and green. Field of vision improved	R. eye no sense of light L. eye + sense of light − projection	Reduced	Standstill for 2 years then deterioration. At discharge he tells of his own accord that he can see the colours of the traffic lights.
III	— 0 Cells 312/3 A.50 G.2	2×8 S. inj. 40° 12×1 S. cc. bism.	—	R. eye movements of hand in front of eye. L. eye counts fingers 25cm field of vision reduced	Ibid. Ibid. Field of vision reduced	+ sense of light + sense of light − projection	Reduced	Worse, vision rapidly declining. On admission in three weeks from 6/24 both eyes to 6/60 R. eye, <6/36 L. eye, the nearly normal red field of vision lost.
IV	2060/00 0 40 Cells 64/3 A 25 G.1	3 S. inj.	Nausea Vomiting Cold shivers Pains	—	R. eye counts fingers 1 m L. eye movement of hand Field of vision reduced	—	—	Treatment not carried through. Does not answer questionnaire.
V	— Cells 183/3	8 S. inj. 39·5°	—	R. eye 5/18 + 1·0 L. eye 5/12 Distinguish red & green	R. eye 5/18 − 1·0 L eye 5/12 Field of vision considerably increased + red and green both eyes	—	—	Improved. Does not answer questionnaire.
VI	— Cells 8/3 A. < 10 G. < 1	8 S. inj. 39°	—	R. eye counts fingers 50cm. L. eye no sense of light + sense of colour both eyes	R. eye 3/24 + 5·0 Sees red Field of vision improved L. eye + projection	—	—	Improved. Correction of eyes not determined at first examinations. Does not answer questionnaire.

TABLE V.—CASES OF ATROPHY OF THE OPTIC

Case No.	Record. Sex. Age.	Years since Syphilis Infection.	Treatment.	Symptoms.	Acuity of Vision.	Condition of Eyes.	Wassermann Reaction (blood) Kahn.
VII	121/33 Male 37	15	—	For 6 months declining vision. ÷KF ÷AF +Ataxia	R. eye movements of hands in front of eyes L. eye 5/6 +1·0	Pupils equal round. +Argyll Robertson syndrome. Discs sharply outlined. Diffuse atrophy	000101 00 0 2400
VIII	82/34 Male 65	21	25 mercury inunctions 5 mercury injections +neo-arsphenamin. Since then no treatment	For 5 years declining vision +Uncertain gait +girdle feeling ÷A.J. ÷K.J. +Ataxia +Romberg	R. eye 5/36 L. eye 5/36 No sense of colour both eyes	Macula cornea Pupils irregular +Argyll Robertson syndrome Discs diffuse atrophy	10·601 00 0 30
IX	170/34 Male 55	Knows nothing about syphilis	—	For 4 months declining vision. +Difficulty with test phrases. ÷K.J. ÷A.J. ÷Ataxia	R. eye +sense of light ÷projection L. eye no sense of colour 5/24 +1·0	Macula on R. cornea. Pupil slightly responds to light and accommodation Ophthalmoscopic R. eye impossible L. eye Disc sharply outlined diffuse atrophy	+D.20 0 6
X	599/34 Female 45	24	Mercury inunctions and "medicine"	For 4 months declining vision +Babinsky Otherwise no symptoms.	R. eye + sense of light L. eye 5/12 Field of vision normal	Pupils respond to light, R. slowly Discs white diffuse atrophy	+D.8 +D.9
	II Admission				R. eye — sense of light L. eye 5/12 Field of vision reduced	Pupils do not respond to light	
	III Admission				R. eye +sense of light L. eye 5/12 Field of vision further reduced		+D.2 +D.2

NERVE TREATED WITH SULFOSIN—*continued.*

Case No.	Spinal Fluid.	No. of S. Injections Maximum Temp. Auxiliary Treatment.	Discomforts.	Acuity of Vision at end of Treatment.	Acuity of Vision 1 year later.	Acuity of Vision at present moment.	Field of Vision	Total Result. Remarks.
VII	— 0 40 Cells 27/3 A.28 G.2	9 S. inj. 39·7° A.T. 10 S. inj. at other hospital	Pains at side after injection	R. eye + sense of light L eye 5/6 Field of vision improved	R. eye + sense of light L. eye 5/6 Field of vision same	R. eye + sense of light L. eye 2/60	Same	Worse. Removed to another hospital on account of dermatitis.
VIII	—	8 S. inj. 39.7°	—	R. eye 5/36 + 2·5 Field of vision improved Both eyes distinguish red 10/300 L. eye 5/18 + 2·5	R. eye Ibid. Field of vision improved Sees red 10/300 L. eye > 5/24 + 2·5	R. eye 5/36 L. eye 5/24	As 3 years earlier	Improvement, then standstill. 3 months after discharge red is seen about 20° to all sides of centre. Sees the colours of flowers in his garden. Green sometimes seen as blue.
IX	00040/ 00 J. Cells 35/3 A.10–12 G.0	8 S. inj. 39 8° bism. 1936 8. S. inj. 40°	+ Herpes + Nausea	R. eye + sense of light L. eye 5/24 – 5/18 Field of vision improved	R. eye Ibid. L. eye 5/18 Field of vision normal + sense of colour	— L. eye 1/60	Worse	Improved, then became worse. 20 years ago acetic acid in R. eye which has since been blind. States that immediately after injection of S. vision declining, to improve later on. During treatment, red 20/300 was seen centrally.
X	30–100 + D.4 + D.1 Cell 83/3 A/30 G.3–4 + D.2 + D.2		A.T. A.T.	— —	— —	— —	— —	I Admission, treated with 9 bismuth and 9 neoarsphenamin- + Sulfosin. Worse. II Admission (15/8/35) A.T. (+ Sulfosin) Worse. III Admission 2/8—23/8/37 + S. Improved.
		8 S. inj. 40·3°	—	R. eye + sense of light L. eye 5/9 Field of vision improved	—	R. eye – sense of light L. eye 5/9 Field of vision same as on discharge	R. eye – sense of light L. eye 5/9 Field of vision better than on discharge	

TABLE V.—CASES OF ATROPHY OF THE OPTIC

Case No.	Record. Sex. Age.	Years since Syphilis Infection.	Treatment.	Symptoms.	Acuity of Vision.	Condition of Eyes.	Wassermann Reaction (blood) Kahn.
XI	220/35 Female 61	28	50 mercury inunctions	For 4 years declining vision, since then A.T. ÷K.J. —A.J. +Aortitis	R eye counts fingers 50cm. L. eye 5/6 + 1·50	R. pupil > L. Do not respond to light, Discs pale R. diffuse atrophy porcelain white. Arterio-sclerotic retina (winding vessels of irregular calibration)	1931 000201 00 80 1935 ÷ +D.4
XII	572 35 Female 45	25	Mercury inunctions 1910 1915 mercury injections	For 6 months declining vision in left eye +headache. Memory declining. No other symptoms.	R. eye 5/6 cm. L. eye counts fingers 1m.	R. pupil > L. Respond to light and accommodation, L. slightly R. disc temporal atrophy. L. disc diffuse atrophy	+D.18 +D.14
	II Admission			The same	R. eye 5/6 L. eye 3/36 ÷ 1·00 Field of vision slightly reduced since I discharge		
XIII	73/36 Male 39	Knows nothing about syphilis	—	For 3 years declining vision in right eye. About one month also left eye. Shooting pains in arms and legs. Tendon jerks extinct. ÷Ataxia +Abadie's sign	R. eye no sense of light L. eye 5/18 + 0·5 Field of vision much reduced pa ticularly for red	R. pupil does not respond. L. pupil responds to light. Diffuse atrophy of R. disc. Temporal atrophy of L.	+D.1 ÷
XIV	461/36 Male 33	Knows nothing about syphilis	÷	For 12 months declining vision. Memory bad. +Difficulty with test phrases. +K.J. slight on right side. +Fibrillation of face. ÷Ataxia. Drinks.	2/60 4/60 both eyes field of vision reduced ÷sense of colour	R. pupil < L. irregular, respond to light and accommodation. Discs greyish white diffuse atrophy Lamina cribrosa visible	+D.5 +D.3

NERVE TREATED WITH SULFOSIN—*continued.*

Case No.	Spinal Fluid.	No. of S. Injections Maximum Temp. Auxiliary Treatment.	Discomforts.	Acuity of Vision at end of Treatment.	Acuity of Vision 1 year later.	Acuity of Vision at present moment.	Field of Vision.	Total Result Remarks.
XI		6 S. inj. 40°	Cold shivers	R. eye movement of hand in front of eye L eye 5 6 + 1 50 Field of vision as at admission	Ibid. Ibid.	—	—	Moved to Medical Department on account of Aortitis. Treatment with S. not continued. Does not answer questionnaire.
XII	+D.4 Cells 24/3 A.16-18 G.0	8 S. inj 39° + bism. & iodine	Slight discomfort	R. eye 5 6 +0·5 L. eye 3 36 +1 0 Field of vision improved	—	—	—	Acuity and field of vision improved. During S.T. discs grey with new blood vessels. During II series of S. the acuity of vision of eye improved to 5/60.
		8 S. inj. 38·6°	Headache & Nausea	R. eye 5/6 L eye 5/60	R. eye 5/6 L. eye counts fingers 50 cm. Field of vision same	R. eye 5/6 L. eye same	Same	Treated strong A.T. between two admissions and later; pupils as in 1935.
XIII	+D.6 Cells 225/3 A.25.30 G.2	8 S. inj. 40·1° 12×1.5 c.c. bism.	Pains in legs after injection	R. eye – sense of light L. eye 5/9 Considerable improvement of field of vision, also for red	R. eye same L eye 5/24 Field of vision same	R. eye same L. eye <5/24	Same as in 1936 perhaps slightly reduced for red	Central vision improved, then it declined slightly. Field of vision improved then standstill. Stated considerable subjective improvement during S.T. Vision was always better 3—4 days after S. R. pupil responds normally.
XIV	— Cells 4, 3 A.8 G.0	8 S. inj. 39·5°	—	R. eye 5/36 L. eye 5/60 Field of vision slightly improved both eyes Sees red 15, 300 both eyes.	R. eye 1/60 L. eye 4/60 Field of vision same No sense of colour	—	—	Improved during S.T. afterwards deterioration and standstill. Does not answer when written to. Has tried to commit suicide.

TABLE VI—CASES OF INTERSTITIAL

Record Sex Age	Eye attacked	Length of stay at Hospital Days	Vision on admission	Deep vessels in cornea	Precipitates Membranes	Wassermann reaction Kahn. Spinal fluid
Rigs Hosp. Dep. H. W. S., Male 24	Both eyes	(77) 22 after commencement of S.T.	?	Many before S.	+P. +P. before S.	40·100 10
Frederiksb Hosp. R. J., Female 20	Both eyes	?	?	Many before S.	+P. +P. before S.	00020100 2400
Rigs Hosp. Dep. H. G. M., Female 4	Both eyes	37	?	Few	—	00010100
587/33 Female 9	Both eyes	53	R. eye 6/12 L. eye 6/6 9 days later P.K.	Many before S.	+P. +P. before S.	000010100 320
56/34 Female 7	Both eyes	(141) 53 after commencement of S.T.	R. eye movement of hand L. eye: 5/6 normal, 6 days later P.K.	Many before S.	– —	0020100
102/34 Female 10	I. R. eye II. L. eye	48 22	—	Few Few	— —	010100
378/34 Male 12	II. R. eye I. L. eye	(45) 26 after commencement of S.T. (163) 101 after commencement of S.T.	R. eye 5/18 L. eye counts fingers 25 cm. R. eye 5/6 (P.K. 1 month after discharge)	Few Many before S.	+P. +P. before S.	+ Spinal fluid W.R + Cells 43/3 A. 10 G. 0

KERATITIS TREATED WITH SULFOSIN

Record Sex Age	No. of days before eyes were white	Vision > 6/18 after S.T. commenced	No. of S. injections Max. Temp.	Relation between A.T. and S.T.	Treatment	Remarks
W. S.	13	?	81 max. 4 c.c. 38·6°-40·7°	First A.T. then S.T.	Local A.T.	Re-appearance of K.P. on R. eye, healed 6 months earlier. Previous A.T.
R. F.	10	?	81	First A.T. then S.T.	Local A.T.	Previous A.T.
G. M.	10	?	91 max. 4 c.c. 38·2 40 0	First A.T. then S.T.	Local A.T.	
587/33	53	80	71 0·4-1 c.c. 37·9-39·8	First S.T. then A.T.	Local Lemon diaphoresis Vitamin D A.T. Arc-light	Temperature reaction bad. Irritation following neoarsphen.
56/34	27	2 years later R. eye: counts fingers 50 cm. L. eye 5/36	81 1-2 c.c. 38·1-40·4	First A.T. then S.T.	Local Lemon diaphoresis J.M. A.T. Vitamin D	Increased photophobia and retinitis following neoarsphen. Infiltration at site of injection.
102/34	46 14	153 76	91 0·5-1 c.c. 38-40·2	First S.T. then A.T.	Local Lemon J.M. Arc-light A.T. Pagenstecher ointment	About 2 months between 1st and 2nd eye.
378/34	(27) 10 after S.T. 17	43 2 years later L. eye 6/60	61 0·5-2 c.c. 38-38·7 81 0·5-1·5 c.c. 38·2-39·7	First A.T. then S.T.	Local diaphoresis A.T. J.M. Arc-light	Previous A.T., at home for syphilitic children 6 months, later iridocyclitis. Vision declines R. eye 6/12, L. eye 6/60 between 1st and 2nd admission.

TABLE VI—CASES OF INTERSTITIAL

Record Sex Age	Eye attacked	Length of stay at Hospital Days	Vision on admission	Deep vessels in cornea	Precipitates Membranes	Wassermann reaction Kahn. Spinal fluid
522/34 Male 20	Both eyes	113	R. eye 5/12 + 1 0 L. eye 5/36 + 1'0	Few Few	+ P. } before S. + P. } before S.	0'20 100 80 Spinal fluid Cells 54/3 A. 40 G. 3 W.R 060100 Kahn 4
421/35 Female 11	Both eyes	(97) 76 after commencement of S.T.	R. eye 5/60 L. eye 5/6	Many Many	+ P. } before S. + P. } before S.	+ D. 11 + D 10 Spinal fluid Cells 0/3 A. 8 G. 0 W.R. +
473/35 Female 22	I. R. eye	71	R. eye counts fingers in front of eye L. eye 5/6 normal	Many before S.	—	+ D. 6 + D. 6
82/36 Female 13	I. R. eye	38	R. eye counts fingers 1 m. (L. eye 5/6 normal)	Few	+ P. } before S.	—
	II. L. eye	31	L. eye ? (R. eye 5/6)	Few	+ P. } before S.	
198/36 Male 9	I. L. eye	44 37 after commencement of S.T.	R. eye 5/6 normal L. eye movements of hand in front of eye	Few	+ P. before S.	+ D. 11 + D. 10
365/36 Female 27	I. R. eye	34	R. eye movements of hand in front of eye 5/6 normal	Many before S.	—	+ D. 9 + D. 8

KERATITIS TREATED WITH SULFOSIN—*continued.*

Record Sex Age	No. of days before eyes were white	Vision > 6/18 after S.T. commenced	No. of S. injections Max. Temp.	Relation between A.T. and S.T.	Treatment	Remarks
522/34	18	About 2 years later R. eye <6/6 L. eye 6/36	81 1-2·5 c.c. 38·8-39·5	First A.T. then S.T.	Local J.M. A.T. Arc-light	Syphil. encephalopathy. ,, choroiditis. Previous A.T. Iridocyclitis glaucom. eye, injection nearly vanished after 3rd S.
421/35	41	I. 2 years later 6/18 II. 35	61 0·5-2 c.c. 38·6-39·6	Simultaneous A.T. and S.T.	Local A.T. J.M. Pagenstecher ointment	Hyphaema disappearing during treatment. Mild case 2nd eye after S.
473/35	71	1 year later 6/60	81 1·5-4 c.c. 39·1-39·7	Simultaneous A.T. and S.T.	Local A.T.	+ Hypopyon. More irritation after neoarsphen.
82/36	19	19	61 1-1·5 c.c. 39-39·6	S.T.	Local Vitamin D.	Previous A.T. Choroiditis syphil. Mild attack in 2nd eye. (+ S. for 1st eye.)
	23	30	61 1·5-2 c.c. 37·7-39·6			Urather. congen. syphil. 3 months between 1st and 2nd eye.
198/36	37	140	81 1-3 c.c. 37·7-39·4	Simultaneous A T. and S.T.	Local Lemon diaphoresis A.T.	3 months after discharge small K.P. on R. eye disappeared with atropine. Vision 5/12 (not admitted), severe photophobia, injection, lacrimation disappeared after 1 S.
365/36	34	Vision clears very slowly	81 1-5 c.c. 37·6-39	First A.T. then S.T.	Local A.T.	Previously A.T., also immediately before admission (cause of the abundant corneal vessels?). Corneal vessels caused slow clearing. Heavy injection and photophobia disappeared after 3 S. injections.

TABLE VI—CASES OF INTERSTITIAL

Record Sex Age	Eye attacked	Length of stay at Hospital Days	Vision on admission	Deep vessels in cornea	Precipitates Membranes	Wassermann reaction Kahn. Spinal fluid
372/36 Male 30	I. R. eye	(35) 31 after commencement of S.T.	R. eye movements of hand L. eye 5/6 normal	Many before S.	+ P. before S.	+ D. 5 + D. 2
387/37 Female 44	I. L. eye	(40) 35 after commencement of S.T.	R. eye 5/6 normal L. eye movements of hand in front of eye	Many before S.	—	+ D. 4 + D. 1
413/37 Male 14	II. R. eye I L. eye	33 62	R. eye II. < 6/6 L. eye 6/24 + 1·0	Few	— —	+ D. 7 + D. 6
487/37 Female 38	Both eyes	(117) 103 after commencement of S.T.	R. eye movements of hand L. eye?	Many before S.	+ before S. +	+ D. 8 + D. 8 Spinal fluid Cells 43 A. 10 G. 0 W.R.+

KERATITIS TREATED WITH SULFOSIN—*continued.*

Record Sex Age	No. of days before eyes were white	Vision >6/18 after S.T. commenced	No. of S. injections Max. Temp.	Relation between A.T. and S.T.	Treatment	Remarks
372/36	31	6 months later 6/36	81 2-4 c.c. 38·8-40·0	First S.T. then A.T.	Local A.T.	Vessels causing slow clearing. Heavy injection of eye and photophobia disappeared (after 1 S. inject on) and the eye was paler. Irritation following neoarsphen.
387/37	35	1 year later movements of hand	71 2-4 c.c. 38·6-40·2	First S.T. then A.T.	Local A.T.	Scaly syphilide disappeared after S. Vision improves slowly (on account of vessels?)
413/37	16 62	33 141	81 2-3 c.c. 39·1-40·8	First S.T. then A.T.	Local Lemon diaphoresis Arc-light Vitamin D.	Vision L. eye decreases during stay at hospital (counts fingers 1 m.). 46 days after S. 6/24. 3 months between 1st and 2nd eye.
487/37	103	(After 46 days 6/24)	14 3-4 c.c. 37·7-40·2	First A.T. then S.T.	Local A.T.	Was immediately given A.T. (Bismuth caused further irritation in spite of S. Abscess at site of injection—incision).

TABLE VII.—CASES OF INTERSTITIAL

Record. Sex. Age.	Eye attacked.	Length of stay at Hosp. Days.	Vision on Admission.	Deep vessels in Cornea.	Precipitates Membranes.	Wassermann Reaction Kahn Spinal fluid.
84/30 Female 14	Previously R. eye	22	R. eye 5/24 – 4.0 R. eye counts fingers 1½ m.			0040100
	II. L. eye			Many	–	160
129/30 Male 18	II. R. eye	65	R. eye 5/18 (L. eye 5/60)	Many	+ P	00100
	I. L. eye	58	L. eye counts fingers 2 m. R. eye 6/6	Many	+ P	+
47/31 Male 19	II. R. eye	33	R. eye < 5/9	Many	+ P	–
	Previously L. eye			Many		–
140/31 Male 9	I. R. eye	51	R. eye movements of hand	Many	–	00040100 320
	II. L. eye	61	L. eye counts fingers 2 m.	Many	–	
189/31 Female 13	II. R. eye	33 } 86	L. eye movements of hand	Few	–	0000
	I. L. eye	52 }		Many	–	320
366/31 Female 20	I. R. eye	(115) for eye 43	? L. eye 5/6 normal	Many	–	–
384/31 Female 10	Both eyes	57	?	Many	–	00020100
			?	Many	–	160
408/31 Female 17	Previously R. eye		R. eye counts fingers 2 m.	Many		+ 40
	II. L. eye	57	L. eye counts fingers 0·5 m.	Many	+ P	On discharge 0·10·60100 160
156/32 Male 31	I. L. eye	40	R. eye 5/6 normal L. eye movements of hand	Many	–	40/00 10
274/32 Female 18	Previously R. eye		R. eye 5/9 + 1·0 ⌒ –1·0 0°	Many (obl.)		–
	II. L. eye	49	L. eye < 5/6 – 0·5	Many	+ P	
382/32 Female 13	I. L. eye	43	R. eye 5/6 normal L. eye counts fingers 1 m.	Many	–	00060100 80
435/32 Female 20	I. L. eye	84	R. eye 6/6 + 4·0 normal L. eye counts fingers 3 m.	Many	+ P	10100 10
448/32 Female 15	I. R. eye	43	R. eye movements of hand L. eye 5/24 + 5·0 ⌒ + 1·0 0°	Many	+ P	000010100 80

KERATITIS NOT TREATED WITH SULFOSIN

Record.	No of days before eyes were white.	Vision > 6/18	Treatment.	REMARKS.
84/30		> 1 year	Local A.T.	5 years earlier similar disease in right eye. R. eye corneal maculae.
	22	4 years		
129/30	32	32	Local Sarsaparil diaphoresis.	3 months between 1st and 2nd eye.
	137	Ab. 7 months		
47/31	33		Local Sarsaparil. diaphoresis	Previous A.T. It is not known when the L. eye was attacked.
140/31	42	83	Local. Sarsaparil. and Lemon diaphoresis. A.T., I.M.	2 years between 1st and 2nd eye.
	60	60		
189/31	31	33	Local. Sarsaparil. diaphoresis A.T. I.M.	2nd eye attacked 1st day after discharge.
	54	69		
366/31	43	>3 months	Local. I.M. Sarsaparil. diaphoresis	Previous A. T. Arthritis, which causes the long stay at hospital. The length of disease of the eye is reckoned till the time the eye grew white.
384/31	240	R. eye 192 L. eye ?	Local. Sarsaparil. and Lemon diaphoresis. A.T.	
408/31	?		Local Diaphoresis I.M.	Anterior choroiditis in both eyes. Re-appearance of disease in R. eye, when L. eye is attacked. 6 weeks between 1st and 2nd eye.
	39	52		
156/32			Local A.T.	Previous A.T.
	124	?		
274/32			Local. Sarsaparil. diaphoresis I.M.	3 years at home for syphilitic children Previously A.T. 7 years between 1st and 2nd eye.
	Ab. 90	> 4 years		
382/32			Local. Lemon diaphoresis, dionin A.T.	A.T. after eyes became white. No irritation.
	29	32		
435/32			Local Diaphoresis I.M. A.T.	
	?	>5 months		
448/32	5	> 3 years	Local Diaphoresis A.T.	Anterior choroiditis in both eyes. opacities of the vitreous body.

TABLE VII.—CASES OF INTERSTITIAL KERATITIS

Record. Sex. Age.	Eye attacked.	Length of stay at Hosp. Days.	Vision on Admission.	Deep vessels in Cornea.	Precipitates Membranes.	Wassermann Reaction Kahn. Spinal fluid.
510/32 Male 11	Both eyes	99	R. eye 5/6 normal (later K.p.). L. eye counts fingers 0·5 m.	Many Many	– –	000060100 320
211/33 Female 12	Both eyes	(20*)	R. eye 5/18 L. eye 5/12	Many Many	+P +P	0060100
248/33 Female 18	I. R. eye II. L. eye	49 42	5/36 5/18	Many Many	+P	020100 80 On discharge 00060100 80
285/33 Female 20	I. L. eye	31	R. eye 5/6 normal L. eye < 5/60	Few	–	–
3 54/33 Male 5	Both eyes	56	?	Few	–	+
252/34 Female 38	Both eyes	(10*)	R. eye movements of hand L. eye counts fingers 1 m.	Few Few	+P +P	– 4
1 67/35 Male 22	Previously R. eye II. L. eye	95	R. eye 5/6 L. eye counts fingers 0·5 m.	Many	+P	020100 9 Spinal Fluid Cells 0/3 A.10 G.0 W.R. –
233/36 Male 9	I. L. eye	(27*)	R. eye 5/6 normal 5/18	?	+ P	+ Degree 2 + ,, 2
471/36 Female 48	Previously R. eye II. L. eye	37	R. eye 5/6 L. eye counts fingers 2 m.	Many	+P +P	+ Degree 1 + ,, 2

ABBREVIATIONS TO TABLES V, VI, VII.

A. - - . Albumen.
A.j. - - Achilles jerk.
A.T. - - Anti-syphilitic treatment.
C. - - - Children.
D. - - - Degree.
G. - - - Globulin.
J.M. - - Syrup of iodine and mercury.
K.j. - - Knee jerk.
K.p. or p.k. - Parenchymatous keratitis.

NOT TREATED WITH SULFOSIN—*continued.*

Record.	No. of days before eyes were white.	Vision < 6/18	Treatment.	REMARKS.
510/32	78 } 79 80 }	73 ?	Local. Diaphoresis I.M. A.T. Arc light	
211/33	?	?	Local Lemon diaphoresis	*Moved to venereal dept., not counted Previous A.T.
278/33	7 30	23 39	Local Diaphoresis A.T.	About 2 months between 1st and 2nd eye.
285/33	?	87	Local. Diaphoresis I.M. Pagenstecher ointment Saline sub-conj.	Previous A.T.
354/33	?	> 3 years	Local Lemon diaphoresis A.T. I.M.	
252/34	?	Ab. 1 year	Local A T.	Dement. 5 months earlier k.p. on L. eye, re-appeared, when R. eye was attacked. *Not counted, discharged at own wish.
167/35	84	46	Local Diaphoresis I.M.	10 years previously similar affection in R. eye.
233/36	24	?	Local Diaphoresis	Moved to Medical Department on account of albuminuria. *Not counted.
471/36	105	161	Local I.M.	11 years previously k.p. in R. eye. Previous A.T.

ABBREVIATIONS TO TABLES V, VI, VII.

M. - - - Men.
P. - - - Precipitates.
S. - - - Sulfosin.
S.T. - - Sulfosin treatment.
W. - - - Women.
I. - - - First attack.
II. - - - Second attack.
P.H. - - Pain on pinching heel.

AN UNUSUAL CONDITION OF THE POSTERIOR SURFACE OF THE CORNEA (POSTERIOR HERPES OF THE CORNEA)

BY

L. STAZ

JOHANNESBURG, SOUTH AFRICA

MR. M. D., when first seen in January, 1932, was aged 18 years. He had had glasses from an optician two years previously. He complained of a pain behind the left eye of about two months' duration, and inability to shoot. The latter complaint, no doubt, was advanced in order to obtain a certificate of exemption from military service.

On examination, the patient was found to be a well-built youth with a bad complexion. His entire face was badly covered with acne vulgaris and comedones. His vision was—

Right =6/9 (ii) Left =6/9

Right with $\frac{+0{\cdot}75 \text{ D. sph.}}{+0{\cdot}5 \text{ D. cyl. Axis } 115^\circ}$ =6/6

Left with +0·75 D. sph. =6/6 most.

During retinoscopy one was struck by a peculiar reflex in the beam of light, and further examination with the loupe, and then with the corneal microscope showed this to be caused by an opacity on the posterior surface of both corneae.

The opacities were well seen with the Zeiss Corneal Microscope Ocular No. 2 and Objective A2 giving a magnification of 23X.

In the right cornea (Fig. 1*a*), in its upper half, was found a longitudinal grey-white area, and in an irregular row in the centre of this band were a few clear circular holes. The band was located in the position of the corneal endothelium and the clear areas caused the slit-lamp beam to bulge into the anterior chamber. They looked like small vesicles, but the posterior surface could not be seen, and one was left with the impression that the clear areas were ruptured vesicles. There was a similar condition in the lower half of the left cornea.

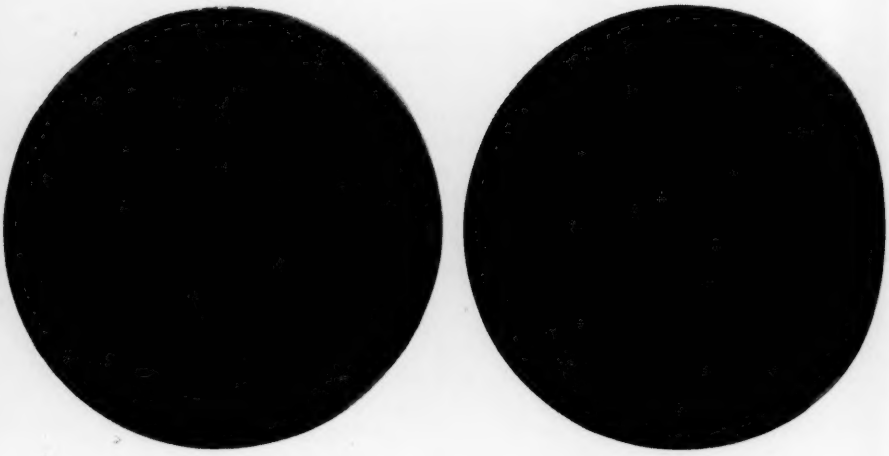
In February, 1933, there was an extension of the condition in both eyes (Fig. 1*b* and 2*b*).

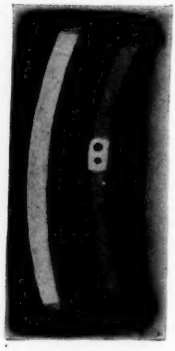
In May, 1933, and January, 1934, no change was noted.

On August 20th, 1934, the condition was as follows:—

In the *right eye* the opacity stretched from above the centre of the cornea to " 9 o'clock " on the edge of the semi-dilated pupil (Fig. 2). The grey area was of uneven thickness, being denser at the temporal end, and the clear areas were punched out, with raised edges. It appeared as if the layer of endothelial cells had become curled forward and folded over.

RIGHT. FIG. 3. LEFT.

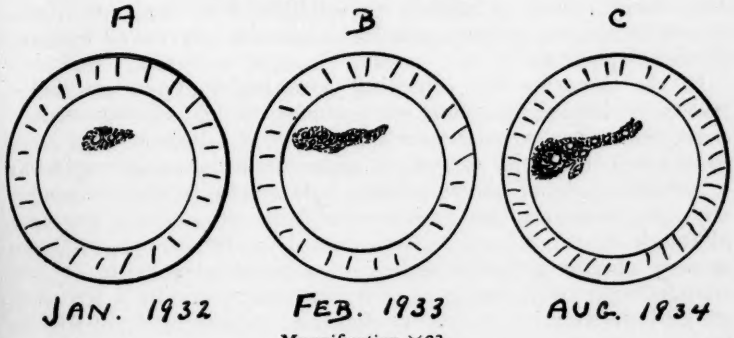




OCTOBER, 1938.

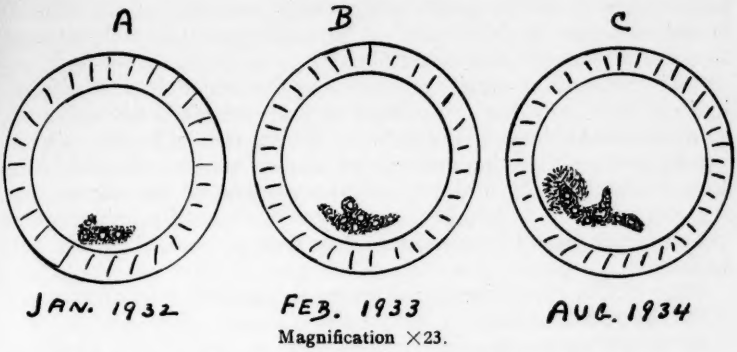
UNIV. OF MICH.

FIG. 1. RIGHT EYE.



Magnification ×23.

FIG. 2. LEFT EYE.



Magnification ×23.

In the *left eye* the condition was situated at " 6 o'clock " on the edge of the iris. The nasal end was surrounded by a nebulous haze containing no holes.

When examined in the zone of specular reflection, the changes were found to have occurred in the endothelium. The corneae were slightly thickened.

Vision without glasses—Right=6/9 partly. Left=6/9. His complexion was as bad as at the original examination. There had never been pain or inflammation in the eyes. He was not subject to the common cold and had never had herpes labialis.

In March, 1936, examination revealed no change in the condition with the exception that in the left eye, the clear gaps were beginning to coalesce in some cases and caused large gaps in the endothelial layer.

In October, 1938, the condition was still as shown in Fig. 3 (drawn from rough diagrams). There was now a suspicion of

early interstitial changes in the right cornea, superficial to the deep area. Vision unaided was—Right=6/9. Left=6/6. In the right eye, the opacity was just visible to the naked eye, on careful inspection.

In the literature the following paragraphs appear in Koby (1930): "Under the name of posterior herpes of the cornea, Schnyder describes a curious condition, of which he has seen three cases in several years. It occurs in people suffering from recurrent attacks of herpes labialis, which seem to become gradually less severe. The patient complains of pain in the eye, although there is no appreciable injection. By focal light, there is seen on the posterior surface of the cornea a more or less rounded superficial opacity, like a thin veil containing a few very stable bullae having a bright reflex and measuring from 0·05 to 0·1 mm. in diameter. If these changes are focussed in the posterior zone of specular reflection, it is found that the bright yellow pattern of the endothelial cells is marked by dark holes in the situation of the bullae. The outlines of the cells cannot be seen in the region of the bullae."

"The superficial opacity surrounding the bullae, their visibility in focal light, and the coincidence of herpes labialis are sufficient to differentiate these changes from Hassal-Henle bodies, which can be seen only in the zone of specular reflection. Kneusel has seen similar vesicles on the posterior surface of the cornea, in four patients who had no herpes. There were usually half a dozen vesicles from 30 to 100 microns in diameter surrounded by a slight haze."

Peter (1932) recognises three types of endothelial dystrophy:—

(1) that observed after iridocyclitis,

(2) following trauma,

(3) true dystrophy of the endothelium.

He states that all cases should be studied by direct illumination along the line of specular reflection. In type 1 are seen a varying number of black pigmented spots lying upon endothelial cells of blurred outline and even surface. In type 2 the changes are usually limited to the region affected. The endothelial mosaic varies in level, oedema is marked and evanescent black spots are seen. The epithelium is usually involved. In type 3 the condition is permanent and bilateral and begins in the centre of the cornea. It can only be recognised in its incipiency by the slit-lamp when large round black non-reflecting areas appear. Vision remains perfect. In the second stage the lesion advances towards the periphery and the endothelial surface takes on the appearance of beaten silver, the hexagonal cells become fewer and a burnished silver network surrounding black non-reflecting areas can be seen everywhere. Vision falls and the fundus is seen with difficulty.

Dystrophy was found in Peter's series in association with senile and nuclear cataract being usually in patients over fifty years of age. The disease constitutes a serious complication in operative conditions probably due to loss of vitality. There is no tendency to regeneration.

Basil Graves (1924) described his observations in twenty-two cases of a bilateral chronic affection of the corneal endothelium of elderly persons. His cases appear to be cases of "cornea guttata" and differ from that now reported.

Goar (1934) summarises his paper on dystrophy of the corneal endothelium as follows:—

"(1) Dystrophy of the corneal endothelium or cornea guttata (Vogt) is a clinical entity occurring in some degree in about six per cent. of people or persons above twenty years of age.

"(2) It usually begins in the fourth or fifth decade, and is three times as common in women as in men.

"(3) It is a chronic slowly progressive disease of the corneal endothelium, resulting in irregular thickenings and excrescences of Descemet's membrane. These excrescences are usually directed posteriorly and have identical staining properties with the membranes from which they are derived.

"(4) Advanced cases of this disease reduce the vision very materially, and these may be recognised by oblique illumination and by the ophthalmoscope. Early cases may be diagnosed only by biomicroscopy.

"(5) Epithelial dystrophy is often found in advanced cases, and it may be the late stage of the process which is represented by endothelial dystrophy as an earlier stage."

In the discussion, various speakers emphasised that in the early stages of this condition of cornea guttata centralis, the corneal microscope is necessary for its detection, and also that it is associated with senile and pre-senile changes. The case which is now being reported occurs in a young man, and the details are easily visible with a loupe and focal illumination.

Freudenthal (1933) describes "Two cases of familial endothelial dystrophy of the cornea with general degenerative changes." In the anterior corneal lamellae delicate nebulae were seen. The posterior surface of the cornea was very rough and in many spots the endothelium was greatly swollen while in narrow contiguous zones the endothelium was vesicular. With lower magnification, these areas looked like sharply circumscribed deposits on Descemet's membrane of a peculiar yellowish white colour. These lesions were uniformly distributed over both corneae. Visual acuity was reduced. The son had myopic astigmatism, divergent strabismus, and general debility, probably resulting from a thyroid-hypophyseal dysfunction. A younger son was

normal generally and had normal eyes. The wife had dementia praecox, his sister a syphilitic lesion of the cornea and her son normal cornea and hyperthroidism.

Juler (1934) presented a man, aged 39 years, with a rupture of Descemet's membrane following contusion as a result of a war injury. The left cornea showed a sinuous line on its posterior surface; it did not reach the limbus. From this, a shelf projected back into the anterior chamber, evidently formed by Descemet's membrane. There was a thinning of the posterior layer of the cornea corresponding to the previous bed of the shelf. In two places there were seen to be some rounded globules growing from the posterior corneal surface in the shelf area.

Doggart (1933) who in recent years has published a good deal of work on the corneal complication of acne rosacea, and other skin conditions, makes no mention of any similar case.

Sholi, of Fuknoka, Japan (1931), has described a pigmented network of the posterior surface of the cornea in a patient 64 years of age, who had a cataract extraction. He concludes that it is the result of an anomaly in the early stage of development of the eyeball.

Cases of Groenouw's nodular degeneration of the cornea have been reported repeatedly—Judd (1933), Treacher Collins (1902), and Marcus Gunn (1902), reported a case showing deposits on the back of the cornea, following irido-cyclitis. They were few in number and worm-like in appearance.

The case reported above is one of " Posterior Herpes of the Cornea "—Kneusel's sub-division, as described in Koby (1930) " Slit-lamp Microscopy of the Living Eye," or the earliest stage of dystrophy of the corneal endothelium.

The patient reports for examination whenever requested to do so. In 1937 he married and now has a child. It is hoped that both he and his off-spring will remain under observation for many years.

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STUDIES ON THE BACTERIOLOGY OF HYPOPYON ULCER

III.—A Bacteriological Investigation of 120 Cases of Hypopyon Ulcer

BY

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Introduction

A SPECIAL investigation into hypopyon ulcer is being conducted in Scotland under the auspices of the W. H. Ross Foundation for the Study of Prevention of Blindness. Certain preliminary observations, on the conjunctival flora of mine workers, who constitute a high percentage of the admissions to hospital for this condition, have already been published (Rhodes, 1938, 1939 *a*, *b*). At present, certain members of the Clinical Staff of the Eye Department, Royal Infirmary of Edinburgh, are engaged in investigating established cases of hypopyon ulcer, or ulcer of hypopyon type. Bacteriological examination of these cases is also being carried out, and in this paper I will present briefly my results in the first 120 cases, 67 per cent. of which occurred in mine workers. It should be understood that this paper is preliminary, and that full ophthalmological details of this series of cases will be published later by the Ross Foundation.

Methods

Material to be cultured is taken, with a small wire loop or metal spud, from under the edge of the corneal ulcer. If the ulcer is small and the edge indefinite, the surface is touched with the loop; in such cases cocaine may sometimes have to be instilled, but this can usually be avoided. Inoculation is carried out on to blood agar medium, in a special screw-topped vial. After suitable incubation, investigation of the resultant bacterial growths follows the usual lines, and in the case of *Pneumococcus* this examination includes typing by mouse injection. At first, typing tests were only carried out to differentiate between Types 1, 2, 3 and Group 4. Recently, however, certain Group 4 strains have been accurately placed in their correct Type by further tests.

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* Working under the auspices of the W. H. Ross Foundation for the Study of Prevention of Blindness, Edinburgh.

Results

The results of the cultural examinations are given in the following table.

Bacteriological Findings in 120 Cases of Hypopyon Ulcer

Result of cultures from edge of ulcer	Number of cases	Percentage of total
No growth on culture	26	21·6
Diphtheroid bacilli	39	32·4
Staphylococcus albus	15	12·5
**Pneumococcus*	14	11·6
Staphylococcus albus and diphtheroids	9	7·5
Staphylococcus aureus	4	3·3
Diplobacillus of Morax	4	3·3
Anthracoid (sporing aerobic) bacilli	4	3·3
Streptococcus viridans	3	2·5
Streptococcus haemolyticus	1	0·8
Staphylococcus albus and anthracoid bacilli	1	0·8

* Nine of the 14 strains belonged to Group 4, and it was further found that two of these belonged to Type 29, and one to Type 28. The remaining five strains proved avirulent to mice, and so could not be typed.

Discussion

A noticeable feature of the investigation is the comparatively high percentage of cases (21·6 per cent.) in which culture yielded no growth, and it would appear that there are at least four possible explanations for this: (*a*) the specimen may have been improperly taken, and inadequate material obtained to inoculate the cultures satisfactorily; (*b*) the particle striking the patient's cornea may have itself set up ulceration and hypopyon, without the necessity for the presence of an accompanying bacterial infecting agent; (*c*) a non-cultivable agent, such as the herpes virus, may have been responsible; (*d*) it is thought that certain corneal ulcerations may be of an allergic nature (see, *e.g.*, Duke-Elder, 1938).

I do not think that technical reasons were primarily to blame for these negative cultures, for such results were obtained from quite large ulcers, where adequate specimens could be readily obtained. With regard to the rôle of the traumatising particle, an interesting observation was made: namely, only one sterile result was obtained in 21 cases of injury due to stone (*i.e.*, 4·8 per cent.), whereas in 50 cases of injury due to coal, there were 14 negative cultures (*i.e.* 28 per cent.). The precise explanation

of this difference is, however, at present uncertain. With regard to herpes virus, it is known that this may be present in the conjunctival sac and give rise to infection of corneal injuries, with the production of dendritic and other types of ulcer. It is possible that it may have been responsible for certain of these cases, although this is, of course, pure speculation.

In all probability, the correct explanation of negative cultures in hypopyon ulcer varies from case to case.

The organisms most commonly isolated from these cases were diphtheroid bacilli and *staphylococcus albus*, either alone, or in combination. It must be borne in mind that the isolation of these organisms from the ulcer may have been due to a purely accidental contamination from the conjunctival sac, where they are known to occur as commensals in a high percentage of normal persons. In other cases, however, it is probable that they played a definite pathogenic rôle, perhaps aided by some special susceptibility of the corneal tissue to infection.

Hypopyon ulcer is frequently spoken of as the " pneumococcal ulcer," and certain workers have found these organisms in 60-70 per cent. of cases (see Duke-Elder, 1938). In this series of cases, however, pneumococci occupy a relatively unimportant place (11·6 per cent.).

Other pathogenic organisms which were occasionally isolated were *Streptococcus viridans, Streptococcus haemolyticus, Diplobacillus of Morax, Staphylococcus aureus*, and anthracoid bacilli. There is little doubt that pneumococci and these other pathogens should be considered as the primary infective agents in the cases where they were isolated.

My observations show that, rather than being a condition of almost specific (*i.e.*, pneumococcal) aetiology, hypopyon ulcer may be associated with a wide variety of organisms. Further, with the exception of *streptococcus haemolyticus* and *staphylococcus aureus*, all the organisms isolated in this series are those that I have previously demonstrated (1939 *a*, *b*) in normal conjunctival sacs. There would not appear to be any valid reason why these two organisms also should not occur in healthy sacs.

It may be said that my investigation tends to lessen the emphasis to be placed on the purely infective element in hypopyon ulcer, for it would appear that although in certain cases recognised pathogenic organisms may be responsible, in others organisms usually regarded as commensals may be isolated. In the latter cases it is probable that the influence of the traumatising particle itself, or the powers of resistance to infection of the corneal tissue, are more essential factors in the causation of the ulcer than the presence of bacteria.

In the pathogenesis of any one case of hypopyon ulcer, therefore, three variable factors must be considered: (*a*) the bacterial

flora of the conjunctival sac of the traumatised eye; (*b*) the nature of the traumatising particle; and (*c*) the resistance of the corneal tissue to infection.

Summary

The bacteriological findings in 120 cases of hypopyon ulcer are presented and discussed. In contrast with previous workers, pneumococci have been isolated much less frequently. It appears that a wide variety of bacteria may be found in cases of hypopyon, while on occasions the infective element is probably lacking or altogether of minor importance.

Acknowledgments.—It is a pleasure to acknowledge my indebtedness to the Clinical Staff of the Eye Department, Royal Infirmary of Edinburgh, for kindly affording access to their cases, and on certain occasions for taking the cultures themselves.

I wish also to thank Dr. A. H. H. Sinclair and Col. R. M. Dickson of the Ross Foundation, Professor T. J. Mackie, and Dr. J. C. J. Ives for help and advice during the progress of this work.

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INFANTILE DACRYOCYSTITIS TREATED BY SURGICAL DIATHERMY

BY

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DURING the past few years, several cases of intractable dacryocystitis in infants have come under my observation and care. In each case, a history could be obtained of neglected epiphora at birth, followed by one or more attacks of acute dacryocystitis, with abscess formation and the development of a lacrymal fistula. All the infants concerned were undersized and undernourished, and no radical operation could be attempted until they had been hospitalised for several weeks and their general condition sufficiently improved to warrant a general anaesthetic being administered.

The usual method of dacryocystectomy produced disappointing results. Many difficulties were encountered, chief among them being the extremely limited field of operation, excessive haemorrhage, and adhesions existing between the sac and the surrounding tissues. Local reaction often caused considerable alarm. One infant developed a well-marked cellulitis, with a sharp rise of temperature to 105° F.: stitches had to be removed, and the wound drained, the condition taking many weeks to settle down.

It was with the object of overcoming such difficulties that I decided to attempt complete obliteration of the sac by the aid of surgical diathermy. The results have proved highly satisfactory.

Under chloroform anæsthesia, an incision is made with a small scalpel commencing about one millimetre above the internal palpebral ligament, and carried downwards and outwards to proportionately the same length as that made in an adult dacryocystectomy. When the incision is being made the fistula must be divided into two along its whole length. The wound is then deepened, the internal palpebral ligament divided, and the sac laid open. The wound cavity is plugged tightly with pledgets of wool soaked in a 1-1000 solution of adrenaline chloride.

The indifferent electrode of the diathermy apparatus is placed under the child's bare buttocks, and the packing removed from the wound. An assistant holds the edges of the wound apart with a pair of narrow sharp-pointed retractors. (The individual parts of a special small-sized Axenfeld retractor, such as I am in the habit of using in adult dacryocystectomies, are excellent for the purpose.) The current is adjusted to an adequate cauterizing strength and directed through a thin blunt-pointed probe into the wound. The interior of the sac is thoroughly cauterized, care being taken to include the infundibulum and the entrance of the nasal duct into the nose. Special precaution must also be taken to prevent scorching of the surrounding skin. Finally the divided halves of the fistula are cauterized. No stitches are required. A small dam of sheet rubber is inserted into the wound and left *in situ* for three days. No local or general reaction follows. The wound heals rapidly by granulation, and generally by the end of three weeks a small, neat scar has formed.

My thanks are due to Mr. F. R. Brown, F.R.C.S.E., Visiting Surgeon, Maryfield Hospital, Dundee, for his advice and practical help.

A ROTATING CROSS-CYLINDER

BY

M. TREE

LONDON

THE astigmic lens or cross-cylinder was first described by Stokes in 1849, but its popularisation and practical application to the methods of subjective testing of ametropia, is due to the insight of that "G.O.M." of American Ophthalmology, Dr. Edward Jackson.

The Jackson type of cross-cylinder is mounted in a fixed position with its handle at 45° to the cylinder axes.

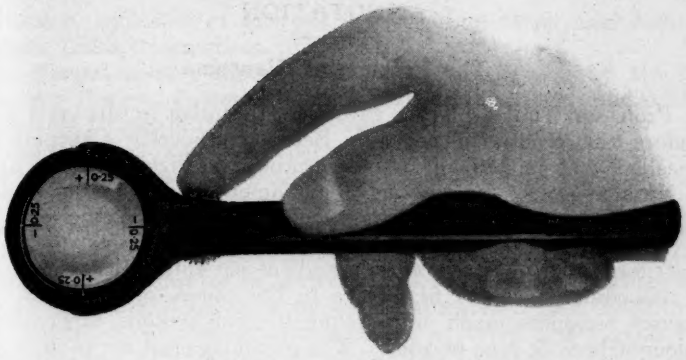
The manipulation of this cross-cylinder necessitates two procedures:—

1. Orientation of the cylinder axes, which implies rotation in a clockwise or anti-clockwise direction, in front of the trial-frame which the patient is wearing.
2. Twirling of the cross-cylinder.

The act of twirling produces a rotation of the cross-cylinder axes through 90°. Dr. Jackson insisted that twirling gives absolute suddenness to the change of direction, and thus produces that maximum contrast which is necessary to help the patient in deciding on the combination which gives clearest visual acuity.

It is strange to note that in spite of the enthusiastic advocacy of the cross-cylinder by the celebrated Dr. Jackson, it is still not used with the frequency that it deserves. To some, no doubt, the action of the cross-cylinder is still wrapped in mystery, but I do not think that this is the entire explanation of its neglect. It has long seemed to me that although the mounting of the cross-cylinder in a fixed position is useful to facilitate the act of twirling, it introduces grave inconvenience where orientation of the cylinder axes is concerned. The act of rotating the Jackson cross-cylinder is apt on occasion to become an awkward procedure, which is irritating both to patient and oculist alike. If the handle is not to abut on the patient's nose, it is equally liable to become entangled in the knobs and adjusting mechanisms of the modern trial-frame.

To overcome these inconveniences, Messrs. Theodore Hamblin have constructed a rotating cross-cylinder, to my specification, which is here illustrated. The handle is mounted at right angles to a semi-circular scale which is marked in degrees from 0° to 180° as on the ordinary trial frame. The cross-cylinder lens is mounted within the semi-circular scale, and is rotated in it by means of a cog-wheel situated at the upper part of the handle.



During orientation of the cross-cylinder, the handle is held vertically in front of the trial-frame worn by the patient. The lens is rotated into its optimum position by means of the cog-wheel, which is turned by the examiner's index finger, and the handle is meanwhile retained steadily in the vertical position. The exact positions, in degrees, of the cross-cylinder axes are at once apparent on the scale. The next step is to rotate the whole cross cylinder lens through 90°—which is the procedure comparable to twirling the original Jackson cross-cylinder. This rotation through 90° may be accomplished in several ways:—

1. The cog-wheel may be used and the lens quickly and suddenly rotated. This is facilitated by the fact that the new position of the appropriate axis is already indicated on the scale, by the position of the meridian of the opposite cylindrical component.

2. Instead of using the cogwheel, the handle may be swung outwards from the vertical to the horizontal position.

3. In the special circumstances, where on orientation, the cylinder axes are at 45° to the handle, *i.e.*, with the axes at 45° and 135° the handle may be twirled as with the Jackson cross-cylinder.

I am quite convinced that by either of the first two methods the cylindrical lens can be rotated through 90° with sufficient suddenness to provide the abrupt contrast necessary. It is true that the act of twirling an orientated Jackson cross-cylinder may give serene pleasure to the joyful oculist and perhaps impart the impression of some magical sleight-of-hand; yet it should not be forgotten that the net effect is simply to rotate a lens through 90°. I venture to suggest that this pleasure is so often outweighed by the preceding difficulties, that the adoption of a rotating type of cross-cylinder, such as I have above described, will by its simplicity and ease of manipulation conduce to the more extended use of the cross-cylinder.

ANNOTATION

The Problem of Glaucoma

Professor Emil de Grósz gave the Hunterian lecture* this year before the Hunterian Society. Before considering glaucoma he recalled that in 1892 a special meeting was held in London to discuss a project for erecting a monument by international subscription to that great Hungarian medical pioneer, Semmelweis.

His lecture reviews the problem of glaucoma. His experience is vast for he has operated on thousands of patients with this disease. "Glaucoma takes the first place in the causes of blindness. It causes blindness much more frequently than syphilis, tuberculosis, blennorrhoea or even injuries." The percentage is lower in countries of high culture with well developed public health administration. The percentage in England and Wales is 9 per cent.; in Holland, 13 per cent.; in Switzerland, 14 per cent.; in Germany, 15 per cent.; in Hungary, 17 per cent.; in Bulgaria 19 per cent.; in Greece, 24 per cent.; and in Egypt, 27 per cent. At de Grósz's hospital in Budapest the proportion of out-patients with glaucoma was 1·4 per cent. and 6 per cent. of in-patients were treated for glaucoma. Glaucoma is especially prevalent among the Jews. In 30 years experience at Budapest 12 per cent. were Jews forming 28 per cent. of the glaucoma patients. During his 30 years directorship of the clinic 5,000 operations were performed. Iridectomy, 2,773; trephining, 755; iridosclerectomy, 168; cyclodialysis, 1,006; anterior sclerectomy, 215; iridencleisis, 10.

The lecture by one of the world's doyens of ophthalmology is of great interest; it would certainly have pleased John Hunter had he been alive to hear it and we hope it will be widely read.

OXFORD OPHTHALMOLOGICAL CONGRESS

The Congress gathered at Keble College on July 5 and on July 6 Mr. Percival Hay (Sheffield) was installed as Master.

The Doyne Memorial Lecture was delivered by Mr. F. A. Williamson-Noble on "The ocular consequences of certain chiasmal lesions."

In the discussion on "orthoptic training" the treatment of heterophoria by this means was critically reviewed. Some emphasized the immense service that this treatment afforded in removing headaches, strains and potential neuroses whilst others maintained that relief from such symptoms could be afforded by a careful

* Published by the Royal Hungarian University Press, Budapest (R. R. Thiering).

refraction, occlusion of one eye and attention to physical health without orthoptic exercises.

A discussion on "Miners' nystagmus" was opened by Mr. T. Harrison Butler. Postural, lighting, toxic, psychasthenic and other associated factors in this disorder were reviewed.

The following is a list of other scientific papers read and discussed before the Congress:— "Ocular filariasis, with a report of a case in which *microfilaria Bancrofti* was seen in the anterior chamber," Mr. W. H. McMullen. "The ophthalmic surgeon, a functional operative paresis," Mr. Basil Graves. "Number forms," Mr. P. G. Doyne. "The aqueous, a secretion," Dr. J. Douglas Robertson. "How is a hyphaema or a hypopyon absorbed?" Mr. Thomson Henderson. "The latest principles for applying tinted glass to industrial and other purposes," Sir Arnold Lawson. "Drusen of the retina: a clinical and pathological study," Dr. Bernard Samuels. "The clinical application of vital staining in fundus lesions," Mr. Arnold Sorsby. "Some successful corneal grafts," Mr. Tudor Thomas. "A new unobtrusive occluder," Mr. F. E. Preston. "Eye injuries—workmen's compensation and disabilities schedules," Sir Arnold Wilson.

Abstracts of some of these papers will be published in this section of the Journal later.

Cinematograph films were shown by Mr. R. Weedon Butler on Lexer's operation, Miss Margaret Dobson on orthoptic training in cases of convergent concomitant strabismus and Dr. La Rocca (New York) on a new method of stitching for corneal grafts.

ABSTRACTS

I.—GLAUCOMA

(1) **Schmelzer Hans (Erlangen). Research on general causes of glaucoma. (Untersuchungen ueber allgemeine Glaukomursachen).** *Arch. f. Ophthal.*, Vol. CXXXIX, p. 465.

(1) **Schmelzer** examined the blood serum of a large number of glaucoma patients and normal control persons for uric acid, blood sugar, bilirubin, indican, cholesterin and the xanthoprotein reaction, and the urine for albumin, sugar and, in a few cases, urobilinogen.

The results showed that there was never any change in the indican and seldom in the blood sugar or uric acid; on the other hand in almost every case of glaucoma a marked increase of the cholesterin content and a positive xanthoprotein reaction as compared with that in the normal patients, and in 20 per cent. of the

former a rise in the bilirubin in the serum. The different types of primary glaucoma showed no appreciable difference in the various tests. The examination of the urine gave negative results.

These increases very probably indicate a certain disturbance of function in the liver as well as slight renal deficiency. It is uncertain whether these general disturbances of metabolism really constitute the ultimate causes, or only play a co-ordinated rôle in the pathogenesis, of primary glaucoma.

As regards treatment the writer suggests, in conjunction with the usual local therapy, the adoption of general measures, such as the avoidance of alcohol and nicotine and in particular food rich in protein and fat. The injection of insulin in special cases—5 to 10 units daily—in order to reduce cholesterin content is also mentioned.

THOS. SNOWBALL.

(2) **Rubino (Padua).—The condition of the cerebro-spinal fluid in glaucoma. (Ricerche sulla pressione e composizione del liquidocefalo-rachidiano dei glaucomatosi, etc:).** *Rass. Ital. d'Ottal.*, November-December, 1938.

(2) **Rubino** recently published some research on the condition of the cerebro-spinal fluid in cataractous patients; now he extends these researches to the glaucomatous and pays special attention to the behaviour of the filtering membrane (barriera emato-liquoriale) between the blood vessels and the cerebro-spinal fluid. This barrier is represented by the choroidal plexuses and the capillaries of the meninges. In this examination, the fluid was extracted from the fasting patient in the morning, and blood taken from a vein at the elbow at the same time. To test the "barrier" he used Walter's "quotient of bromine" which involves the estimation of bromine in the C.S.F. and the blood; in the latter the concentration is three times as great as in the former.

As a result of the comparison of normal and glaucomatous people, the author finds the calcium in the blood and C.S.F. slightly less in the glaucomatous, but the difference is so little that he considers it negligable. He finds "Walter's quotient" reduced in glaucoma, which shows an increase of permeability of the haemato-liqourial barrier. This fits in with modern theories of glaucoma.

HAROLD GRIMSDALE.

(3) **Casini (Pisa).—A case of chronic glaucoma with delayed hypertension. (Considerazioi e ricerche in un caso di glaucoma cronico semplice a ipertensione tardiva).** *Arch. di Ottal.*, January, 1939.

(3) **Casini's** case was that of a woman, aged 66 years, who had noted slight visual failure for a year without pain until ten days

before her visit, when for the first time she had violent pain about the left eye. The vision of the right eye was 4/10; there was slight cupping of the disc; the left eye had a turbid vitreous and the fundus could not be seen. The visual fields were concentrically contracted to 15°. Various tests were made.

Increase of fluids in the diet was followed by marked diuresis. Injection of histamine was followed by a great increase of the intra-ocular pressure of the right eye, and a less rise in the left. The action of histamine is a dilatation of the capillaries, together with a vaso-constriction of the arteries. The rise in glaucoma seems to depend on the increased permeability of the capillary system. It is probable that in the case under examination there was a sudden rise in pressure in the left eye, giving rise to the pain of which the patient complained; afterwards this pressure passed oft, leaving the tension lower than normal.

HAROLD GRIMSDALE.

(4) **Nicolato (Pavia).—The aetiology of glaucoma following division of after-cataract. (Note sull'etiologia e la patologia del glaucoma conseguente a discissione di pseudo-cataratta).** *Arch. di Ottal.*, January, 1939.

(4) A patient who had undergone cataract extraction without any complication, after division of the subsequent after-cataract, developed acute glaucoma which eventually yielded to iridectomy. A few days later the patient died from cerebral haemorrhage. **Nicolato** discusses the cause of the glaucoma. He comes to the conclusion that the increase of tension must have been due to oedema of the vitreous. Examination of the eye showed no adhesion of the iris or capsule to the wound, nor any swollen lens débris in the vitreous. When the vitreous is markedly degenerate, the eventual result is usually good. The final cause is probably to be sought in a variation of the state of the vessels of the uvea and retina.

HAROLD GRIMSDALE.

(5) **Friedenwald, J. S. and Stiehler, R. D. (Baltimore).—Circulation of the aqueous. A mechanism of secretion of the intraocular fluid.** *Arch. of Ophthal.*, November, 1938.

(5) **Friedenwald and Stiehler's** work was undertaken with the aim of discovering the character and mechanism of the irreciprocal permeability of the ciliary body, which allows fluid to pass with ease from its capillaries to the posterior chamber but obstructs the passage of fluid in the reverse direction. The experiments were carried out with acid and with basic dyes and the following conclusions were reached:

1. Under normal respiratory conditions, acid dyes are transferred from the epithelium to the stroma and basic dyes from the stroma to the epithelium. The phenomena disappear under asphyxia and can be accounted for by an ionic current carrying anions from the epithelium to the stroma and cations from the stroma to the epithelium.

2. By study through indicators of the oxidation and reduction processes in the ciliary tissues it was shown that the potential of the epithelium in air was 0·100 volt, while that of the stroma in air is -0·130 volt. Under nitrogen (acid p.H.) or in the presence of cyanide this difference of potential was abolished.

3. It could be accounted for, at any rate in part, by the presence in the epithelium of indophenol-oxidase and the absence of this enzyme in the stroma. Hence the abolition of the potential difference by asphyxia.

4. When this potential difference is abolished, the stroma-epithelial barrier shows a selective permeability in favour of acid and neutral dyes and a relative impermeability to basic dyes. If the acidity is raised still higher, the selective permeability is reversed and this point is known as the iso-electric point. From data such as the width of the iso-electric zone the potential generated by the oxidation-reduction system is calculated with certain assumptions to be 0·140 volt at p.H. 6·6. Consequently, electrons can be received by the barrier from the stroma and transferred to the epithelium.

5. Experimental perfusion of rabbit's eyes with electrically charged solutions shows that the difference of electrical mechanism responsible for the transfer of dyes in the ciliary body is adequate to explain also the transfer of water in this tissue.

6. The ciliary capillaries are much more permeable than either the capillaries of the iris or the stroma-epithelial barrier, the latter being the least permeable of the three anatomical barriers between the blood and the aqueous. This mechanism must not be thought of as solely responsible for the formation of the intra-ocular fluids.

F. A. W-N.

II.—TRACHOMA

(1) **Foley and Parrot (Algiers).—Sur les formes d'évolution de Rickettsia trachomatis.** *Archiv. de l'Institut Pasteur d'Algérie*, Tome XVI, No. 3, 1938.

(1) **Foley** and **Parrot** have previously reported on the Halberstaedter-Prowaczek Körperchen, the infecting agent of trachoma, which appears as elementary bodies which have their life-history in

the epithelial cells of the conjunctiva. They call them *Rickettsia trachomatis.* (*Arch. Instit. Pasteur d'Algerie*, t. XV, No. 3, 1937, pp. 339-349.) They recognized Lindner's initial bodies, whence are derived the elementary bodies.

The method adopted for obtaining microscopical preparations was as follows:—A fragment of conjunctiva is removed from the everted upper lid near the outer angle; this is spread on a glass slide and fixed in iodized alcohol for four or five minutes, and then stained with a mixture of one drop of May-Grunwald, one drop of Giemsa and 10 drops of neutralized water. Differentiation is carried out with 95 per cent. alcohol.

The initial body stains a deep violet-blue and is 1 or 2μ in diameter; it may occupy any position in the cytoplasm; often two bodies are in close proximity. It increases in size, becoming mulberry-shaped, and divisions becoming apparent; soon it is seen in close proximity to the nucleus of its epithelial cell host.

The elementary bodies are formed after the disintegration of the initial bodies, and appear as dark bluish-violet dots which increase in size to 0·2μ, and become purple in colour. There is a multitude of these tiny bodies which are contained in a fine membranous envelope. This sac may displace the cell nucleus to some extent. It then bursts and the elementary bodies become scattered throughout the epithelial cell, and finally destroy it. At the same time their colour changes, and they become a greenish-blue.

It is to be supposed that the elementary body penetrates into another epithelial cell, and gives rise to a new initial body, and then the cycle recommences.

A. F. MACCALLAN.

(2) **Cuénod and Nataf (Tunis).—Nouvelles recherches sur le virus trachomatoux.** *Archiv. de l'Institut Pasteur de Tunis*, Tome XXVII, No. 3, September, 1938.

(2) The discovery of Rickettsia-like bodies in trachoma was reported by Busacca in the first instance and independently by **Cuénod** and **Nataf** about the same time. The importance of the louse either as a reservoir or as a vector of the trachoma virus has been claimed by the two last-named workers. Poleff has published results of researches on the culture *in vitro* of these Rickettsia-like bodies.

The identity of the Rickettsia-like bodies with the Halberstaedter-Prowaczek Körperchen has been asserted by Thygeson, by Cuénod and Nataf, and by Foley and Parrot. The authors have produced trachoma in man by inoculation with material from lice infected with trachomatous material.

Now the authors state that by inoculating the chorio-allantoic

membrane of eggs with trachoma virus containing the Rickettsia-like bodies they have been able to transmit these bodies from egg to egg during several passages. They also suggest certain histopathological analogies between trachoma and typhus fever.

According to experiment 305 a fragment of trachomatous conjunctiva was received in Paris two days after it had been removed from a patient in Tunis. No growth was obtained on eggs but inoculation of the testicle of a guinea-pig resulted in the development of Rickettsia-like bodies. Another guinea-pig similarly inoculated gave a similar result; with material containing the Rickettsia-like bodies the testicles of rats were inoculated; from these rats the conjunctiva of a Macacus rhesus was inoculated, which resulted in a temporary follicular conjunctivitis of one eye, which the authors consider to have been an attenuated form of trachoma. Therefore the trachomatous virus was conserved in passage through both guinea-pig and rat.

It is known that when the tunica vaginalis of guinea-pigs is inoculated with typhus material epithelial inclusions of elementary bodies (Halberstaedter-Prowaczek Körperchen) are found just before Rickettsiae appear, but are no longer found when the Rickettsiae are at their maximum of development. The authors suggest that these are different stages in the development of the same micro-organism. Analogous observations have been made in trachoma; further research on these lines is proposed.

A. F. MACCALLAN.

(3) **Poleff, L. (Institut d'Hygiene du Maroc).—Passage des Corpuscles Rickettsoides du trachome sur le tissue de l'oeil humain hors de l'organisme et quelques observations generales sur la culture des dites formations in vitro.** *Rev. Internat. du. Trach.*, p. 79, April, 1939.

(3) At the present time it appears to be certain that in trachoma certain very minute bodies are present which have been called "Rickettsia-like," and are generally considered to be the aetiological factor of the disease.

Poleff was the first to have success in the tissue-culture of these Rickettsia-like bodies (*Arch. of Ophthal.*, p. 882, 1936). Here he describes his latest methods in order that the details of his research may be repeated by others.

Outside the organism a growth of the Rickettsia-like bodies is manifested at first only in cellular cultures of conjunctival or corneal trachoma tissue, which has never been treated. This tissue must be obtained by excision and not by scraping, and sown in Carrel's flasks by the authentic methods.

Differentiation of virus particles from the detritus requires

experience but is quite possible; and their microscopy may require enormous patience.

Smear preparations should be made as thin as possible avoiding all unnecessary damage to the cells. "Impression" preparations are advised. Staining should be carried out with weak Giemsa for 24 hours with differentiation by alcohol and xylol. Castaneda-Lepine and Victoria-blue stains are also useful.

In order to effect "passages" on fresh tissue, which is uncertain except with human material, direct and prolonged contact is required. This can be effected by placing the minute fragments of tissue containing the virus on the surface of the new fragments destined for the passage, or by plunging such fragments for half-an-hour in a virulent emulsion before passing to the new culture medium. The best culture medium has been found to be the human placenta.

However, the infection of man from a tissue culture of trachoma has not yet been effected.

The following experiment is described:—A young Moroccan, who was shown by slit-lamp examination to be entirely free from trachoma, required operation for a staphyloma of the cornea. A part of the cornea which was fairly transparent was used for cultures *in vitro*. At the same time tissue was removed from the conjunctiva of another young man with untreated trachoma in the stage II*a*. Tiny fragments of the staphylomatous cornea were placed in contact with pounded up trachomatous material in Tyrode's solution. Five of these fragments were explanted in a hanging drop in a mixture of fowl plasma, human blood-serum and fowl embryonic extract; five in concave slides with human serum (two parts) and Tyrode's solution three parts. Half the cultures were placed in an incubator at 37 degrees, and the rest in an incubator at 32 degrees. As controls cultures of uncontaminated cornea, and cultures of trachomatous cornea were employed.

The result of the experiment was as follows:—After incubation for five days there was an insignificant growth in the plasma cultures, and no change in the liquid medium. By smears no Rickettsia-like bodies were found; by culture on ordinary media no bacteria were found.

After 10 days a distinct increase of Rickettsia-like bodies was found in the infected corneal cultures, and in the trachomatous tissue; there was none in the non-infected corneal culture. Passage on a placenta medium of the Rickettsia-like bodies obtained from the corneal culture was positive.

It therefore seems to the author that corneal tissue, especially if obtained from human embryos, or from corpses immediately after death, forms the best culture medium in experimentation of this nature.

A. F. MACCALLAN.

(4) **Taborisky, J. (Palestine).—Sur les problemes du trachome et les methodes de leur solution.** *Rev. Internat. du Trach.*, April, p. 65, 1939.

(4) It is **Taborisky's** experience that trachoma begins acutely in persons who have never been in contact with any source of contagion up to the time when such contact occurs and when they succumb. When, however, the disease begins insidiously it will be found that the patient has been in continual association with trachomatous persons, a reiterated minimal infection having produced a relative immunity.

It has been asserted by Morax, MacCallan, Wilson and others that it is impossible to differentiate microscopically between trachoma and a follicular conjunctivitis, however, the author is not of this opinion.

A. F. MACCALLAN.

(5) **Cavallacci (Pisa).—The treatment of trachoma by salts of gold. (L'auroterapia nel tracoma).** *Arch. di Ottal.*, December, 1938.

(5) Gold salts were first used in the treatment of tuberculous disease of the eye; after the first expectation (that in gold a specific for tubercle had been found) was disappointed, the salts were used to improve the general defensive power of the organism. **Cavallacci** has found no mention of use in trachoma and has thought it interesting to test it. He used an oily solution of solganol B at first in strength of 2 per cent., and later in 20 per cent.; one series of experiments by instillation into the conjunctival sac; a second series was treated by intramuscular injection of the solution.

The results of the first series were negative; no improvement followed in any case. The second series showed that alone, the drug was not capable of effecting a cure, but when combined with the usual local treatment the disease was more rapidly relieved. The intramuscular injections are well borne.

HAROLD GRIMSDALE.

CORRESPONDENCE

CILIA IN VITREOUS AS A RESULT OF PENETRATING WOUND OF THE EYE

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

DEAR SIRS,—On Wednesday, April 12, a small boy, aged eight years, was struck on the right eye by a pellet from a shot gun.

The eye was seriously damaged; X-ray showed the foreign body to be in the globe; in fact two foreign bodies. (1) The pellet. (2) A much smaller opaque substance which was not found at operation.

The eye was not removed until April 18 as there was some delay in obtaining permission from the parents. Immediately after removal, I opened the eye and found the vitreous infected. The pellet was found in the vitreous and close to it, and seemingly attached to it, were two cilia. The presence of the cilia in the vitreous is unique in my experience, but although it has been noted, it is sufficiently rare to make it worthy of being recorded again.

Yours faithfully,

W. A. ANDERSON.

BELFAST,
July 26, 1939.

OCULAR DOMINANCE IN RELATION TO DEVELOPMENTAL APHASIA

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

DEAR SIRS,—May I draw your attention to points arising out of the review you published of my monograph "Ocular Dominance in relation to Developmental Aphasia" in the current number of your Journal.

The account given there by your reviewer is an exceedingly inaccurate one as well as misleading, and one must conclude that he was able to give this quite sincere piece of work only very cursory perusal before writing his notice.

I shall take up the inaccuracies of statement one by one as they occur.

1. I am attached by your reviewer to the Physiology Department of Edinburgh University, whereas I am on the staff of the Psychology Department—this would give change of direction of emphasis to the investigation described.

2. It is stated that "reading disability in 383 children" is investigated, whereas it is only incidence of disability within this total group that is investigated.

3. It is stated that the mean Intelligence Quotient of this group of 383 children is 100 with probable normal range of intelligence, whereas these findings refer to a smaller group of 49 children who show disability and handicap and are within the larger group.

4. The abbreviated form for Reading Accomplishment Quotient is misquoted as R A, a non-existent term in mental measurement.

These are probably more trivial points, but

5. A rather serious misstatement is made in paragraph 3 of the notice which begins "Only four disability children . . . ." Here two statements made, pp. 48, 30, in quite different reference are brought together, and further, the second of these statements has been misquoted. My own statement (p. 30) is that left-eyedness and reading disability run in families, not left-handedness as your reviewer has it. He therefore has created himself the absurdity to which he draws attention.

6. The final paragraph is, of course, irrelevant to the research and its findings, and arises out of a misunderstanding as to the sense in which the term "ocular dominance" is to be understood in this monograph. On p. 17 especial emphasis is given to this—"It must be emphasised that all three tests (*i.e.*, the tests of eyedness used in this research) are sighting or directional tests of eyedness. In view of the width of the term 'ocular dominance' it is essential to be clear in what sense we will use this term with reference to our findings."

Again on p. 28 the reader is reminded of the nature of the tests used.

Dr. Sampson's finding as to dominance (p. 59) refers solely to tests for retinal rivalry, and no correspondence of results is to be expected from tests by which quite different characters are explored. Put more definitely no correspondence IS found between results from tests for retinal rivalry and from sighting or directional tests for eyedness. Dr. Sampson himself warns the reader to avoid such confusion.

Dr. Sampson examined 36 of the 49 disability cases, and his negative finding is of extreme importance being as it is in complete keeping with what, in consideration of our diagnosis of "developmental aphasia," we had hoped he would find was the case.

The issues involved are not noted by your reviewer, and hence his notice will fail to gain the attention of readers likely to be interested in the monograph. These might very briefly be mentioned.

The diagnostic value of left-eyedness for a very specialised pattern of language difficulty which would appear to be sex influenced.

A new outlook on the problem of severe backwardness in our schools, the aphasic pattern of difficulty not incidental causes or low mental capacity creating the whole "backward" situation.

The interesting genetic situation underlying developmental aphasia and which is now being more adequately explored; I refer here to the finding that heavy incidence of twinning exists in families in which the aphasia occurs.

This makes its contribution to investigation and understanding of ground causes for cerebral sidedness and laterality problems. The important facts remain as Ocular Dominance (understood as defined in this monograph) pointing to issues of cerebral sidedness,

which in turn gives rise to developmental aphasic characters arising out of disturbance of mental functioning.

Before publication the monograph had the perusal and appreciation of men of high standing in the field entered by the research.

The review as published will certainly tend to repel rather than to attract interest for this research and its findings.

I can only see in it suggestions that here is a monograph somewhat slight, somewhat obvious, and in fact fairly considerably stupid.

I would therefore beg to lodge this truly sturdy protest.

Yours faithfully,

A. MACMEEKEN.

PSYCHOLOGY DEPARTMENT,
UNIVERSITY OF EDINBURGH.
August 8, 1939.

The Reviewer regrets the errors pointed out by Miss MacMeeken with regard to "Physiology" and "R. A." (which should of course be A. Q.), and also to the quotation from p. 30, which, as Miss MacMeeken rightly points out is meaningless as recorded, and should run "left-*eyed*ness and reading disability run in families, and in such families there is heavy incidence of left-handedness." He had no wish to "repel interest for this research," and he hopes that both the monograph and Miss MacMeeken's comments will be read and studied.

NOTES

Corrigendum et Addendum

Dr. CARL HAMBURGER, whose paper appeared in the August number of this Journal, requests that the part of the title referring to the author's town be changed to "formerly Berlin, temporarily Geneva." He also wished the following footnote to be inserted:—"This article was completed by the end of 1938, the author who lived in Germany until July, 1939, had no library facilities afforded him in which to give consideration to recent publications in other countries on this subject."

* * * *

The 75th Anniversary of the Foundation of the American Ophthalmological Society

WE have received from Dr. Harry Friedenwald a small brochure giving an account of the origin and first meeting of the American Ophthalmological Society. This is a reprint of an article by Dr. H. D. Noyes, of New York, the Recording Secretary. It was first printed in 1875, and was reprinted for the commemorative meeting in June last. The Society is a very old foundation. It took its rise in the first place by means of correspondence between Hasket Derby, of Boston, and Drs. Noyes and Bumstead, of New York.

On January 9, 1864, a meeting was held at New York in which eight prominent American ophthalmic surgeons were present. Dr. Sands was chosen president and Dr. Noyes secretary. The question of founding an ophthalmic journal was mooted but was not accepted. A committee was appointed to interest ophthalmologists in the States in the new venture, it was composed of Drs. Bumstead, Noyes and Hasket Derby. On June 7, 1864, 18 members met at the New York Eye Infirmary and a committee to draft constitution and by-laws was appointed. On the next day Dr. Dix, of Boston, read papers on what appears to have been a case of pearl cyst in the anterior chamber and on the effects of calabar bean in paralysis of the accommodation following diphtheria, specimens of calabar bean were exhibited. The date of the next annual meeting was fixed for June, 1865, in New York, and a committee on publication elected.

Such was the origin of the American Ophthalmological Society 75 years ago. It may be of interest to remind readers that the Ophthalmological Society of the United Kingdom took its rise in informal meetings in the House Surgeon's room at the old Moorfields Eye Hospital.

Both Societies are getting on in years, both are hale and hearty and we all hope that the vigorous veteran of the States will continue its long and honourable career for many years to come.

* * * *

Société Européenne de Chirurgie Structive

THE Fourth International Congress of this Society will be held in Paris from October 5 to 7, 1939. The Hon. President is Professor P. Sebileau. A discussion will be held on the "treatment of destructions and deformities resulting from scar retraction of the eyelid." The treatment of deformities of the jaw will also be discussed. All communications are limited to subjects coming within the scope of constructive and plastic surgery.

Further information can be obtained from the Secretary, Maison de Chirurgie, 9, rue de Turin, Paris.

* * * *

National Society for the Prevention of Blindness

THE Annual Congress of the Society will be held in New York from October 26 to 28, 1939. The headquarters will be the Astor Hotel. A tentative programme gives communications on Nursing as it relates to sight conservation; sight conservation in industry; the doctor in conserving sight in the pre-school child; social work in prevention of blindness; sight-saving classes. A general summary will conclude the business. There will be a subscription dinner in the evenings of October 26 and 27.

A full syllabus can be obtained from the office of the Society, 50, West 50th Street, New York.

Appointment

MR. R. W. PAYNE has been appointed Hon. Ophthalmic Surgeon to the Plymouth Royal Eye Infirmary.

* * * *

International Council of Ophthalmology

THE International Council of Ophthalmology met in London at the Royal Society of Medicine on April 19, 1939. Present were Messrs. Nordenson (Chairman), Ehlers (Hon. Secretary), Pflüger (Hon. Treasurer), Paton, v. Grósz, Cavara, Terrien, MacCallan, Bailliart, Löhlein, Knapp, Tooke and Sinclair.

The Treasurer reported that the assets of the Council amounted to 2.232 Sw. frcs. The President announced that the German government had agreed to take over the responsibilities undertaken by the former Austrian government with regard to the next Congress. Professor J. Meller, of Vienna, was nominated President of the next Congress. The following themes were chosen for the discussions:—

1. Plastic operations on the eyeball.
2. The pathology of retinal detachment, including the biology and pathology of the vitreous body.

It was decided to lay the question of internationalizing the determination of the axes of astigmatism before the next Congress. The next meeting of the Council was to be held in Paris, 1940, on the day before the opening of the Annual Meeting of the Société Française d'Ophtalmologie.

FUTURE ARRANGEMENTS

1939

October 6.—Midland Ophthalmological Society, at Birmingham and Midland Eye Hospital. (Annual.)

October 20.—North of England Ophthalmological Society, at Manchester.

November 17.—North of England Ophthalmological Society, at Bradford.

December 15.—North of England Ophthalmological Society, at Leeds.

1940

February 2.—North of England Ophthalmological Society, at Newcastle-upon-Tyne.

March 1.—North of England Ophthalmological Society, at Liverpool.

April 5.—North of England Ophthalmological Society, at Sheffield.

July 4—6.—Ophthalmological Congress, at Oxford.

CONTEMPORARY OPHTHALMIC LITERATURE

Archives of Ophthalmology. *July, 1939.*

MARTIN. Cancer of the eyelids.
KIRWAN. Transplantation of the cornea: report of a perfect result.
GORDON. Oculists and occultists: Demonology and the eye.
KRAUSE and STACK. Citric and malic acids of the ocular tissues.
ROSENTHAL. Changes in angioscotomas associated with the administration of sulfanilamide.
GIVNER. Episcleral ganglion cells.
BAILEY and SASKIN. Treatment of severe corneal ulcer with sulfanilamide.
BERLINER. Lipin keratitis of Hurler's syndrome (Gargoylism or dysostosis multiplex): clinical and pathologic report.
LAMBERT. Paget's disease with angioid streaks of the retina.

Annales d'Oculistique. *June, 1939.*

VIALLEFONT, HARANT and TEMPLE. Ocular euliasis.
DRACONTAIDIS. The inversion of the visual field limits for colour in quinine amblyopia.
TISCORNIA and ORTIZ. Trachoma before the law of accidents at work.

July, 1939.

DIEFFENBACH. Tuberculosis of the optic nerve and papilla.
STROOBANTS and SCHEPENS. Acetylarsan and its effects on the visual apparatus.
ADAMANTIADIS. Disciform degeneration of the posterior pole of the retina.

Klinische Monatsblätter für Augenheilkunde. *July, 1939.*

SCHRECK. The clinical and pathological anatomy of orbital tumours.
MEYER. Recklinghausen's neurofibromatosis and the eyes.
PASCHEFF. New investigations about plasmoma conjunctivae.
JAENSCH. Double-eye. Remarks on the work of Dr. Weckert.
GUIST. The loss of visual function from damage to the retina.
TATAR. Retinitis pseudonephritica and periarteritis retinae associated with erythema nodosum.
BOROS. Bilateral macular coloboma in Mongolian idiocy.

Annali di Ottalmologia. *April, 1939.*

BORSOTTI. Paralysis and contracture of the vertical motor muscles of the eye.
SANTONI. Retinal compensation after interruption of the retinal circulation.

Bollettino d'Oculistica. *May, 1939.*

BENCINI. Clinico-histologico-therapeutic considerations on six cases of sympathetic ophthalmitis.
ORZALESI. Respiration and glycolysis in cataract.
PERSICHETTI. Experimental research on the reaction of the eye to the intravenous injection of animal carbon.
SCHUPFER. Research on the behaviour of the light sense in Basedow's disease especially in relation to carotene and vitamin A.

Rassegna Italiana d'Ottalmologia. *March-April, 1939.*

BORSOTTI. The importance of the recognition of the overaction of the inferior oblique in the surgical treatment of paralysis of the superior oblique.
GRANCINI. Meningioma of the orbit.
PERGOLA. A case of herpes of the cornea associated with Claud Bernard-Hörner's syndrome.
AGNELLO. Toxic amaurosis from filix mas.
CARDELLO. Contribution to the study of corneal herpes and disciform keratitis.

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All original communications should be addressed to the Editors, 24, Thayer Street, London, W.1

ANNOUNCEMENT

Theodore Hamblin, Ltd. beg to inform Surgeons that it is not their intention to transfer their business to an address outside London for the period of the War, but to continue to carry on at their present addresses in London as well as at their provincial branches. It is their sincere desire that their service to customers should in no way suffer as a result of the War, although it will be realised that there are a great many difficulties to be met with and overcome.

It may be opportune at the present time for customers to satisfy themselves that they are in possession of all the necessary spare pairs of spectacles they are likely to require. As records are kept of all previous transactions there will be no difficulty in supplying requirements through the post.

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